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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health. The plan to continue the publication of abstracts indefinitely will make the Journal a fund of extensive information, readily available to students, teachers, researchers, and clinicians.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, those papers of scientific and clinical interest which are presented at the annual meetings of that society. Abstracts of important articles related to periodontology that appeared during the year of 1946 will be published in this year's issues.

We are grateful to all who have made available the subject matter from which the abstracts and copies of abstracts have been made, including the authors of the original articles, owners of the journals mentioned in the text of the abstract section, and those who have aided by contributing the material for this issue.

Extracts from an address on

Our Common Battle for the Patient *

By WILLIAM HALE, M.D.,**

. . . "The battle is one *on behalf of* the patient, against disease and disability, waged by us together, in a common cause, and in the ways indicated by our differing kinds of experience. . . .

"We do not question . . . that public health is purchasable; but we do not believe that private health is also purchasable in the sense that money alone can buy it. . . . There are many elements that affect the health of an individual besides the medical care received, and wholly apart from the quality of the public health procedures of the community. . . . A very important one is a coöperative patient with the willingness and ability to follow the advice as to his conduct and behavior which the physician gives. And whether or not the patient follows this advice may depend on the kind of relationship established between him and the physician.

"I am sure it will sound strange for a factual and realistic man who knows himself to be such to stand before you and say that this relationship is primarily an emotional one. Or to add to this statement the additional one that the quality of this emotion is a vitally important factor in treatment. It is a fact which is demonstrated in any doctor's practice every day, that patients who believe in him, who like him, who have sought him as *the* doctor they want above all others—get well faster and more often than those who have no faith. Also, it is a fact that the doctor has an emotional feeling, too, toward the patient who has picked him above all others as *the* doctor for him. . . . The doctor responds to the trust placed in him with a heightened sense of responsibility, and something flows between the patient and the doctor and back to the patient again which can never be analyzed or isolated; never identified, labelled, packaged, and prescribed. But it is there just the same, and every doctor knows it and a great part of his art is in cultivating it and using it for the benefit of the sick person who appeals to him for aid. This is why the doctor knows that mass medicine is not going to work if all or part of this emotional feeling of the patient toward him is deflected to an administrator, or agency, or just to the "government". When the time comes, if it ever does, that people go to a certain doctor because they live in his district, or because they don't have to pay him directly, or because they have a right to demand care . . . then something has happened to that relationship. . . .

"So perhaps there really is a battle going on for the patient after all—a battle for the patient's affections. It is waged by certain enterprising persons on behalf of a government eager to receive gratitude for benefits it has not given and never can give. We dislike it because we think it is basically dishonest, and because we feel we are entitled to have these emotions flow to us, who have earned them, and because we know that when they do so, and when we respond to them as most of us do, they are converted into a healing force."

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* (Delivered before Annual Conference of Health Officers and Nurses, Saratoga Springs, New York, June 25, 1946)

** President, Medical Society of the State of New York

THE JOURNAL OF PERIODONTOLOGY

JANUARY, NINETEEN HUNDRED FORTY-SEVEN

Traumatic Occlusion and the Rest Position of the Mandible

By BERNHARD GOTTLIEB, M.D., *Dallas, Texas**

INTRODUCTION

THE UNDERSTANDING of traumatic occlusion is of great clinical importance. However, the fact that an explanation of periodontal pathologic changes as a result of traumatic occlusion has been misinterpreted in many instances, is not generally recognized. Consequently, tooth grinding as an important phase of periodontal therapy has been introduced. Also bite raising techniques have been developed without consideration for the causes of the original bite closure; may we not then assume that the same causes will remain active and again close the bite of the treated case?

Traumatic occlusion as a factor in full dentures is a neglected subject. The change from the natural dentition to an artificial denture resting as a saddle on the ridge is accompanied by a complete change in the principles of force transmission. The periodontal fibers of the teeth are subjected to traction in function; as long as they can meet the requirements of the applied forces, functional balance is achieved. As soon as the applied forces on the teeth exceed the suspensory capacity of the fibers the force destroys a section of the periodontal membrane, and the pathologic condition of traumatic occlusion is established. The intermediate step of a breakdown of the suspensory capacity of periodontal fibers does not exist with saddle construction such as in full dentures. Here the direct pressure on the bone will tend to produce trauma more quickly than in the natural dentition. Here also pressure on the bone is present right from the beginning.

I shall define the pathologic trauma in both cases caused by pressure on the natural teeth as well as on a saddle. As long as this pressure compresses the connective tissue lining the bone, only to the extent that it is induced to resorb the bone, the pressure may be considered physiologic. We call it "Frontal Resorption" (1). But as soon as this lining connective tissue is compressed to a degree that it is damaged beyond the capacity of performing resorption, a pathological situation exists. The bone at the damaged connective tissue must be removed by the surrounding connective tissue. We call it "rear resorption." This differentiation of physiologic and pathologic results we may encounter in the case of natural teeth as well as under saddle constructions.

It is apparent that there are many facets to the problem of the application of

**Baylor University College of Dentistry*

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excess pressure on teeth in function. An understanding of fundamentals and an analysis of the known facts is the key to a number of clinical problems.

LITERATURE

Wallisch (2) was the first to mention the physiologic rest position of the mandible. He described it as that position of the mandible wherein all muscle action is eliminated and the mandible passively suspended. He reports that in this position the opposing teeth do not contact each other. Fick (3) explained this by stating that the tone of the depressor muscles is greater than that of the elevators. This view is hardly tenable since the muscles which bring the mandible into opposition with the maxilla are known to be far more powerful than their opponents. Fick also reported that the intermaxillary distance of the rest position of the mandible increases as the head is bent backward.

Schwarz (4) showed that the dorsal position of the head produced an opening and distal movement of the mandible while the ventral position caused a mesial and closing movement of the mandible. Thompson (5) confirmed that the rest position of the mandible maintains a space of several millimeters between upper and lower teeth. Nieswonger (6) stressed the advisability of keeping the mandible in the rest position, i.e. with the natural teeth apart, "this permitting the teeth and investing tissues to repair themselves."

Gottlieb (7) called attention to the fact that we use our teeth in function less than one hour a day. Then, if we keep our opposing teeth out of contact at all times between meals (at least 23 out of the 24 hours) we automatically eliminate traumatic occlusion, in producing damage, as a subject for discussion. "We produce traumatic occlusion by pressing the upper and lower teeth together between meals as a result of habit or nervousness." Gottlieb and Orban (8) showed that this pressure results in necrosis of the periodontal membrane between tooth and bone, involving extensive repair. The first one who described rear resorption was C. Sandstedt, a Swedish writer.

Grohs (9) showed that not only can extensive bone resorption be demonstrated under full dentures but also necrosed areas at the borderline between connective tissue and bone. These areas are similar in appearance to those described in the periodontal membrane (8) and are due to the same cause, namely undue pressure. Orban and Weinmann (10) examined a great number of human jaws histologically and found signs of traumatic occlusion in at least one tooth in a jaw in a majority of the examined specimens.

Gottlieb (11) and later other authors have reported pieces of cementum torn away from their connection with the root. The force necessary to produce such a condition must have been great. The periodontal fibers were more firmly connected to these pieces of cementum than were those pieces attached to the neighboring sections of the root of which they originally were a part.

FINDINGS SHOWING DESTRUCTION OF THE PERIODONTAL MEMBRANE IN HUMAN TEETH

The following figures demonstrate various degrees of damage to the periodontal membrane of human teeth which were found in routine examination. When a section of periodontal membrane has been damaged to such an extent that no cellular life exists, the overlying bone must be resorbed before normalcy can be re-established. Frontal bone resorption from this periodontal space is no longer possible since the tissue here is necrotic. The intact connective tissue of adjoining sections of periodontal membrane, sometimes assisted by the connective tissue in neighboring marrow spaces, becomes active in removing the involved piece of bone and then the necrotic tissue. This is the only way the periodontal membrane may be restored to normalcy.

Figure 1 is a section of the periodontal membrane of a lower right second premolar of a person 48 years of age. The lower molars had been missing. The crowns of the upper left canine and premolars, and that of the right first premolar were destroyed by caries and not restored. Thus the only articulating occlusal surface was that of the two right second premolars, which were certainly overloaded. There is a compression of the periodontal membrane so that the nuclei of the connective tissue have been lost and this portion is necrotic. Osteoclasts from the neighboring periodon-

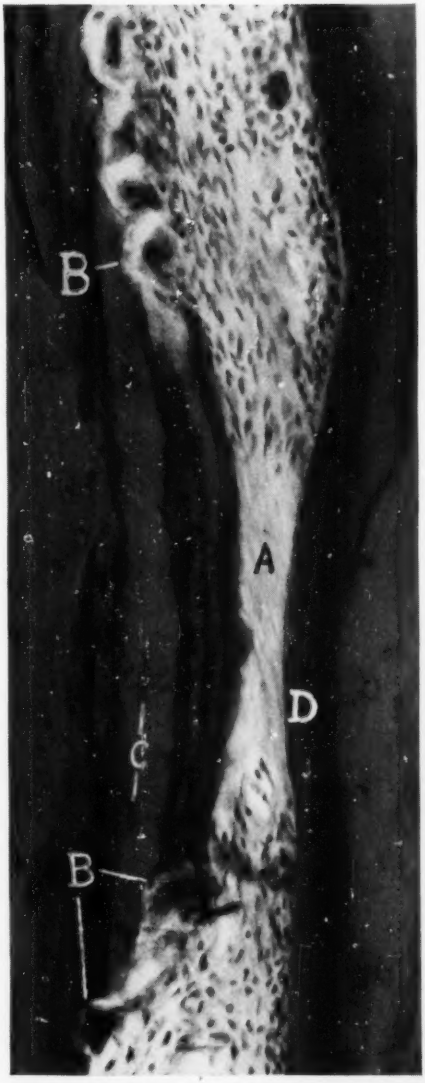

Fig. 1. Periodontal membrane (A) without nuclei, necrotic, (B) osteoclastic rear resorption, (C) osteocytes in bone under resorption, (D) cementum.

tal membrane on both sides have begun to remove the bone in contact with the necrotic connective tissue. Note that the osteocytes are present in the lacunae everywhere, the bone being resorbed even in the immediate neighborhood. This finding is repeated many times.

Figure 2 is a section of the periodontal membrane of a left upper second molar of a person 19 years of age. Lower left first molar and upper right first molar crowns were destroyed by caries and not restored. The other teeth were intact. In the illustration, part of the periodontal membrane is nothing but necrotic debris. Most of the bone has been removed by rear resorption. Numerous round and flat osteoclasts are at work. In one place a narrow splinter of bone is still in contact with the necrotic area of the periodontal membrane. Osteocytes may be seen still present in the bone in the immediate neighborhood of the lacunae. Some lacunae containing osteocytes are opened on one side by resorption. These osteocytes are apparently about to be released into the neighboring connective tissue. The claim that the osteocytes disappear before resorption seems to be based on a misunderstanding. Their presence or absence has nothing to do with the process of resorption.

Fig. 2. (A) necrotized periodontal membrane, (B) last remnants of the bone removed by rear resorption, (C) bone with osteocytes, (D) cementum.

Fig. 3. (A) Place of destroyed periodontal membrane, (B) rear resorption of bone, (C) cementum.

Figure 3 is a different area of the same tooth as Figure 2. Part of the periodontal membrane has been destroyed. Rear resorption is proceeding on both sides, above and below, in areas where the osteocytes may be seen in the bone lacunae. In Figure 2, the process is reaching its end, while Figure 3 shows the process of rear resorption beginning. We may assume that eventually extensive areas of a periodontal membrane may be destroyed, and repeated pressure may prevent many of these areas from undergoing repair. Such a case represents an instance of constant and permanent damage to the periodontal membrane by traumatic occlusion.

Figure 4 shows a completed rear resorption. It is from the left lower second molar of a person 47 years of age, who died of cerebral apoplexy. The four first molars were missing, second and third molars were in occlusion. The two incisors, the cuspid, and the second bicuspid were missing on the lower left side. On the surface of the root is seen an extended necrotic strip which represents necrosed periodontal membrane between bone and tooth. The contacting bone has been resorbed, the tooth remaining intact. Some osteoclasts are still at work removing debris. The necrotic strip may be removed too, or else covered by new cementum formation in contact with the cementum of the tooth around the necrotic material (8). The newly formed cementum will again become attached to the bone opposite by means of fibers.

In deciduous teeth we regularly see destruction of areas of the periodontal membrane between deciduous tooth and bone. The length of the deciduous root decreases, while the masticatory stress increases as the child grows. This produces a disharmony leading to traumatic occlusion with destruction of parts of the periodontal membrane between root and bone (Fig. 5). In two places we see necrotic material instead of periodontal membrane.

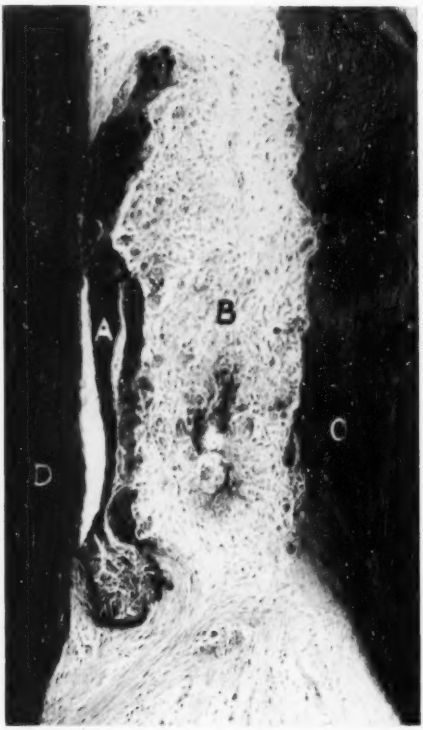

Fig. 4. (A) necrotic periodontal membrane freed by rear resorption, (B) new periodontal membrane, (C) bone, (D) cementum.

As long as the destruction takes place at a distance from the epithelial attachment the immediate danger is not great. Repair may take place at any future time. But if the destruction occurs in the neighborhood of the epithelial attachment, the denuded root surface may be overgrown by the epithelium and the periodontal membrane loss is permanent. That is the salient result of a horizontal component of overloading.

Attention must be called to the fact that the contact of epithelium with forcibly denuded root surface is not sufficient to cause downgrowth of the epithelium all along the denuded surface. If that were the case the flap operation for pocket formation would have been abandoned long ago. To a varying extent it seems always to do this. The quality of the denuded root surface, of which we know so little, is decisive (11).

Figure 6 shows horizontal overloading in a dog's tooth. The periodontal membrane at the alveolar margin has been destroyed. Rear resorption has started to remove the affected bone. The epithelial attachment to the enamel is seen nearby. If this epithelium starts to grow down and the denuded tooth surface has the requisite properties, overgrowth by epithelium will frustrate any attempt at repair. Formation of an effective cementum barrier by new cementum deposition cannot be accomplished as long as healthy connective tissue is not in contact with the cementum surface.

Figure 7 shows the result of holding a pipe between the teeth in a man aged fifty. The teeth subjected to this habit are overloaded to a varying degree, the stress being kept below the breaking point of the pipe stem. No comparison can be made from Figure 7 regarding the level of the alveolar margin on the right and left sides, because the gingiva was peeled off of one half of the jaw and left in place on the other half.

Figure 8 is a sagittal section through the labial pocket of the upper central with the pipe hole. There is a resorption of the tooth apparently caused by holding of the pipe. When it was reached by the epithelial attachment, the latter grew over the resorbed area. The resorption can no longer be repaired by the connective tissue which has been permanently separated from it. Once a surface of tooth is overgrown by epithelium

Fig. 5. (A) deciduous tooth, (B) dissolved enamel of the permanent germ, (C) alveolar bone, (D) necrotic periodontal membrane.

it is eliminated from further connective tissue repair. That illustrates the danger of the horizontal component in overloading.

Figure 9 is the same as Figure 8, except it shows an apical section. There are many excementoses running in the direction of the periodontal fibers. These cementum hyperplasias represent a reinforcement of the fixation of the root. It is known that a lateral component of pressure on a tooth yields greater movement at the alveolar margin than at the apex. The apex is fixed but not the crown. The load of holding the pipe produced an outward tipping component of force. This horizontal component produced the damage at the alveolar margin, resulting in tooth resorption and in stimula-

Fig. 6. Dog—(A) destroyed periodontal membrane at the alveolar margin, (B) rear resorption of the bone, (C) tooth, (D) enamel dissolved by decalcification.

tion of the formation of cementum exostoses in the apical portion of the same tooth.

The preceding illustrations demonstrate damage on the pressure side of the tooth. We usually expect to see no damage on the traction side, and orthodontists assume this correctly. However, a number of observations on human teeth show where cementum pieces have been torn out on the side of traction. In some of these cases it is possible that trauma in the form of a sudden impact may have produced this result. The first published observation of such torn pieces of cementum (12), was in a case of extreme overbite. It was described on the labial at the alveolar margin of the lower incisors apparently due to the horizontal component of the hard hitting, overlapping upper teeth.

Figure 10 shows one of numerous observed cases of torn out pieces of cementum of human teeth on the side of traction. The largest piece is torn out of the dentine. The two smaller pieces are displaced into the periodontal membrane. It is a lower central from a patient aged 47 years. We doubt that such could happen in orthodontics even with the strongest available forces.

TRAUMATIC OCCLUSION

The teeth are suspended in the socket by the periodontal fibers in such a way that force exerted against the tooth will normally be translated into a traction force. Wherever possible, nature always translates force against bone into traction as the most favorable manner of handling it. However, if the force exerted against the teeth is greater than the load the suspending fibers can bear, the tooth is forced into compression of the periodontal membrane, the highest degree being contact of the tooth with the bone. The intervening periodontal membrane is destroyed to a greater or lesser degree.

Can this excess pressure, whatever its origin, be eliminated, or even mitigated by grinding? With certain exceptions, the answer is, definitely not.

Suppose we change the shape of the occlusal surface so that the direction of pressure is changed. The only difference will be that a different site of periodontal membrane will be destroyed. Actually we have made no change in the over-all picture.

Suppose we grind the tooth entirely out of occlusion. The load is simply shifted to the other teeth. In addition, the exarticu-

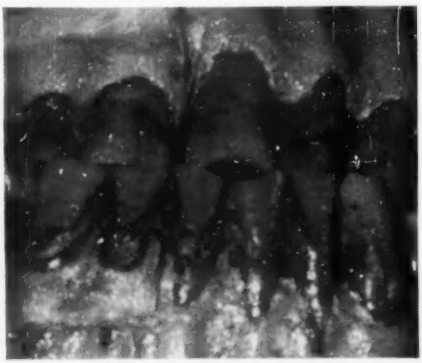

Fig. 7. Pipe hole. Other teeth in end-to-end bite.

lated tooth soon returns to occlusion and the original condition obtains again.

The only reasonable position from which the so-called adjustment of the occlusion may be predicated is in the elimination of horizontal, or forces which act at an angle to the long axis of the tooth. In the molar and premolar region this is not a major consideration since grossly any occlusal force acts axially—minute details do not change the picture materially. In the anterior region it becomes a greater factor than is generally realized.

In the ideal cases the overbite of the anterior teeth tends to wear down to an end-to-end bite. In some cases the overjet may remain. If the tendency of the mandible to anterior movement is strong, and no concomitant wearing down of the overbite develops, the continuous pressure of the upper incisors will cause a crowding of the lowers. It would seem to follow, therefore, that a tendency toward an end-to-end bite is desirable, and the smaller the overbite the greater the possibility of an eventual grinding in to an edge to edge bite. An axial direction will then be given to all forces directed against these teeth. That is also the ideal position for counteracting the continuous active tooth eruption (13).

Constant pressure on the molars and premolars in contact at times other than for chewing at meals, is to be avoided, especially as a result of nervous habits and long chewing of gum. This tends to increase the anterior overlap to a degree that will prevent its elimination by normal wear or by grinding.

The elimination of this overlap by grinding has proved useful when proceeding in the following way: With the teeth in centric occlusion, place the free end of the fingernail against the labial of the lower incisor, the flat of the nail resting against the incisal edge of the upper central. On slow opening, the nail remaining in contact with the lower incisor indicates the amount of overbite to be removed. Its removal is divided equally between upper and lower teeth, and if small in amount may be done with a wheel stone. The incisal edges are then naturally shaped and smoothened. It

Fig. 8. Labial side of the upper central of Figure 7. (A) gingival margin, (B) cementum, (C) dentin, (D) epithelium growth over tooth resorption.

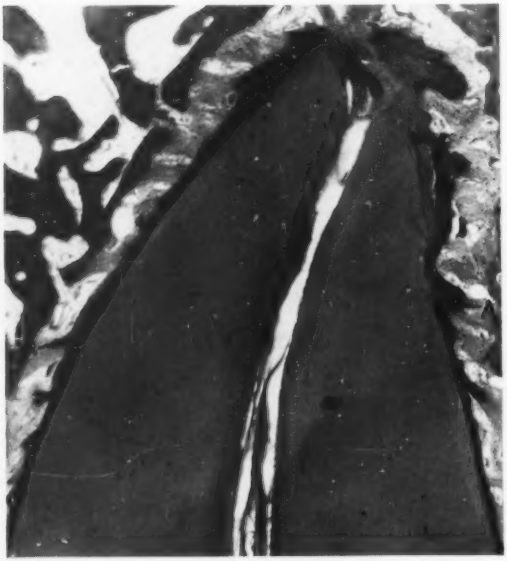

Fig. 9. Apex of tooth of Figure 8. (A) excementoses following the direction of the suspending fibers.

is assumed that the situation has been carefully explained to the patient and his agreement obtained in order to avoid subsequent damage claims.

Esthetic considerations generally militate against the grinding down of anterior teeth especially in women. Years ago their front teeth were normally much shorter, yet their appearance was not less attractive. And from a functional standpoint, it is obvious that the shorter the extra-alveolar arm of the lever, the better. As we tend to eliminate the overbite we tend to eliminate the most harmful component of force against these teeth—the horizontal component.

If the overbite is of such extent that it is beyond correction by grinding, orthodontic treatment is necessary. A bite plate is constructed to be worn at night only. Giving the teeth rest by day allows the periodontal membrane to recuperate from the night pressure and possible damage. In rare cases it may be necessary to cement bands on the upper anteriors with rests for the lower teeth to secure the desired result. In these cases, relief from soreness may be obtained by the patient's bringing the teeth out of occlusion. Soreness indicates contact between tooth and bone, involving damage to the periodontal membrane.

Grinding has a reasonable basis for application in those cases of overloading caused by the wandering of a tooth due to deep cementopathia (14). Such a tooth, suffering from some cementum pathosis, may be ground out of occlusion, giving the connective tissue of the periodontal membrane a chance to restore normal conditions. Sometimes this treatment works and the tooth becomes firm, but *not always*. In many cases, the tooth is soon found in traumatic occlusion again and the wandering continues. Repeated grinding produces the same result and eventually the tooth is exfoliated. *The pathologic wandering is primary, the traumatic occlusion secondary.*

A tooth which is in traumatic occlusion as a result of pathologic wandering will not necessarily be lost if it be not relieved by grinding. It is noteworthy that pathologic wandering is found most often in the upper anterior teeth. There is no plausible explanation for the apparent susceptibility of these particular teeth to *deep cementopathia*. They are the only ones normally able to move freely labially and occlusally with little interference by the occlusion. All other teeth in a well-aligned arch will tend to be held in position by the occlusion.

Excellent results have been obtained in numerous cases by orthodontic means in straightening these pathologic wandering teeth. It would seem that the trauma of forced movement back into the original position produces additional resorption of the tooth surface. With repair following, it is obvious this is a specific means of therapy. Unfortunately, repair does not always follow, and the tooth may be exfoliated.

If wandering premolars or molars are forced back by the articulation in the same manner as orthodontic movement would produce, the same course of events might ensue, i.e., additional resorption followed by cementum deposition. The commonly observed cases of considerable repaired resorptions on the apical root surface may be produced in part this way.

When a portion of the periodontal membrane has been destroyed in such a way that the removal of bone must be effected by rear resorption, the process may take weeks for completion of the repair. Constantly new areas may be destroyed before the old ones are repaired. (Figs. 2, 3.) It may happen that a periodontal area adjoining an epithelial attachment is destroyed, denuding this area of tooth surface. It is probable that the epithelial attachment will tend to grow down, with a consequent elimination of this area for any periodontal repair. It may then be observed clinically either as gingival recession or as pocket formation (14). The resorption areas are sometimes mistakenly diagnosed as caries (Fig. 8).

The reason we do not find these conditions of damage to the area of the alveolar margin more frequently is that the forces exerted on the tooth are not always directed toward the area of the alveolar margin, but rather more apical in direction. Such damage at a point distant from the epithelial attachment is not irreparable and may be restored to normal at any future time by removal of the contacted bone by rear resorption and the deposition of new cementum.

This condition, especially if accompanied by extreme sensitiveness to percussion or even to touch may be misinterpreted with unhappy sequelae for the tooth. Lateral abscesses in teeth sensitive to percussion have been mistaken for necrotic pulps with periapical abscesses. On extraction, the error is not always caught. Opening into the tooth beforehand will reveal sensitive dentine and electric pulp test will reveal a pulp very much alive. And if a lateral abscess cannot be found, the cause of the condition may well be a traumatized periodontal membrane, possibly produced by heavy grinding and pressure of upper against lower teeth especially at night. It is apparent that this knowledge has great clinical significance. Grinding of such a tooth out of occlusion will be of definite help. Numerous teeth have been extracted due to lack of proper diagnosis of these conditions.

Instead of destruction, more gentle pressure on a tooth may produce a widening of the periodontal membrane by bone resorption. Repair may take place by bone deposition or surplus cementum deposition or both. Reduction of the enlarged periodontal membrane at least partly by cementum deposition is preferable since a beneficial increase in root surface results. The additional cementum deposition may follow the course of the suspensory fibers (Fig. 9). The reduction of width of periodontal membrane by cementum deposition in the apical region is especially advantageous since this increases the length of the clinical root, adding to the functional stability. While it is always desirable for a broadening of the periodontal membrane to be followed by cementum deposition, local factors cannot be induced

Fig. 10. (A) cementum torn away from root surface, (B) dentin, (C) cement pieces dislocated into the periodontal membrane, (D) bone.

Fig. 11. Crowns on the second premolar and first molar with soft rubber at the occlusal keeping the other teeth out of contact.

to produce this result as far as present knowledge goes. The whereabouts of the control center which governs cementum formation is unknown. Some connection with pituitary function has been found (15). Ovarian dys-function and cementopathia have been linked, backed by clinical observations. It is possible that the intimate relation of the ovaries and pituitary may be a factor.

Apparently we cannot harm the supporting structures of the teeth during ordinary chewing of food. The thorough mastication of any kind of food produces a force well within the normal limits of the periodontal fibers. Some sections of periodontal membrane may be compressed occasionally, but it is probable that complete rest between meals will allow recovery. However, grinding and pressure of lower teeth against uppers when food is not being masticated, is done with great force and over prolonged periods. This is especially true in nocturnal bruxism. It has been suggested that the poisons from the autointoxication of constipation, circulating in the blood, have a selective action and produce a contraction of certain muscles of mastication. In children some pediatricians ascribe this condition to worms. Sometimes considerable crown loss in deciduous teeth due to grinding is considered pathognomonic.

THE PHYSIOLOGIC REST POSITION OF THE MANDIBLE

The usual concept of the human dentition, natural or artificial, is that of the upper and lower arches in centric occlusal contact. Because of this, we have unthinkingly drifted into accepting it as the normal rest position. This may explain the overemphasis given the relationship of the opposite teeth in centric occlusion.

However, it has been established beyond question that the physiologic rest position of the mandible involves no contact whatsoever between upper and lower teeth. Every investigator in the last 40 years agrees on this without exception. In addition, reason dictates a rest position with the opposing teeth out of contact. There are some people who seem to prefer to keep the teeth in contact in the rest position, though no force is exerted on them. Perhaps a more positive feeling of stability is necessary for these people.

Since contact is the functioning position of the teeth the rest position would involve absence of contact. Absence of contact is necessary to give the supporting structures rest between meals. *The teeth are used at most a total of one hour out of the twenty-four for chewing meals. No damage*

Fig. 12. Bone under full denture. Necrotic area (A) freed by rear resorption, (B), (C) new bone deposition. Courtesy: R. Grohs.

can result from such a short period of use if the remaining rest periods are maintained faithfully.

A careful explanation by the dentist should suffice to make daytime compliance by the patient complete. Further steps may be necessary in those cases where the normal rest position is not attained at night. It might be helpful to mention again the statement from the literature that stretching the neck tends to keep the teeth apart and acquiring this position in sleep may be advisable (4).

DEVICES TO ELIMINATE TRAUMATIC OCCLUSION DURING THE NIGHT

Nocturnal bruxism is difficult to overcome and it is sometimes necessary to resort to splints.

At the beginning of the era of "Traumatic Occlusion" (1890) rubber caps were constructed for a premolar and a molar so that the remainder of the teeth were out of contact (Fig. 11). Concentrating the whole force of the occlusion on two teeth can hardly have sense. A case in point is illuminating. After using a cap like this faithfully every night for some time, one patient observed that the teeth which wore the cap were no longer in contact during the day and she had to replace the cap in order to be able to use the teeth for chewing. This showed that night pressure alone was sufficient to depress the teeth out of occlusion. When she came to me for consultation after her own dentist could suggest nothing to correct the condition, I recommended that she discontinue the use of the cap with the tentative prediction that the teeth would resume their former positions. After several months they had returned to their normal occlusal contact. The patients who were not persistent in wearing these caps did not develop a depression of the teeth.

Experiences of this kind induced the construction of splints over the molars and premolars, both sides being connected with a lingual bar. The result was a depression of these teeth while the exarticulated anteriors continued their active eruption to a severe overbite. The same end may sometimes result from over-indulgence in chewing gum for long periods. A splinting of the teeth appears to be useful only if all the teeth of both jaws are included.

Fig. 13. Bone under full denture. Necrosis at the surface of the bone (A). (B) rear resorption. Courtesy: R. Grohs.

The following technique is used. A continuous splint over all the crowns is constructed of vulcanite or acrylic on a model of plaster of Paris. The occlusal plane of each splint is covered with pink wax, and replaced in the mouth. The patient grinds in his own individual articulating plane and the cases are then cured again, replacing the wax with acrylic or vulcanite. The splints should be used every night. The splints perform a two-fold function. Keeping the teeth apart reduces the total pressure the muscles are capable of exerting. In addition, the pressure is divided among all the teeth and no one group of teeth takes more than its share. This eliminates the ill effects of nocturnal bruxism. Numerous clinical experiences proved it.

TRAUMATIC OCCLUSION AND RAISING THE BITE

The habit of grinding and pressing the teeth together between meals produces such ill effects as to lead to the development of bite opening techniques suitable for the natural dentition. Considerable portions of the crowns may be worn away. As an extreme we are all familiar with the picture of the tobacco chewer with the loss of the entire clinical crown. Yet no food or tobacco is hard enough to wear down enamel. Tooth enamel is one of the hardest substances known and only the friction of enamel against enamel will give a possibility of such excessive wear. It is probable that this happens on one side while the other side is chewing the tobacco. If dentine is exposed, it will be worn down more quickly by the opposing enamel, with resulting groove formation.

Studying the deleterious results of the horizontal component of force on teeth might suggest that axial pressure is beneficial. This is only partly true. Bone resorption in the fundus of the alveolus is caused by tooth depression. This will not necessarily save the tooth, since only aggravating components are eliminated. Ground down teeth even in tobacco chewers are known to become loose and lost. People usually drop the habit when this occurs. The failing of the continuous cementum deposition *(Cementopathia)* is decisive. (14)

We sometimes come across the statement that teeth cannot be depressed orthodontically. It is difficult to understand why this assumption is made. In the use of constant pressure exerted by arches and ligatures, there is the danger of strangulation of the pulp at the apical foramen. But in using a bite plane for depressing the lower anteriors for example, the pressure is relieved every time the mouth is opened. Experiments on dogs (7), cementing crowns on opposing molars produced a considerable depression which was readily apparent in their relative position to the mandibular canal in the lower teeth. Not a single necrosis of the pulp occurred in about 50 dogs. It may be taken for granted that any tooth can be depressed when the force is acting approximately in an axial direction. Resorption of bone takes place wherever pressure is applied either at the alveolar margin or at the apex.

The two conditions, wearing down of the crowns and the apical movement of teeth may sometimes call for raising the bite. It is, however, more often advisable to place occlusal inlays to save exposed dentine from further wear. Technical details may be omitted. Attention is called to the fact that raising the bite, regardless of technique, will fail if the patient is permitted to continue his habit of pressure and grinding between meals. Porcelain restorations will be shattered and metallic restorations will force the teeth to be depressed and in a relatively short time the vertical dimension will return to its original extent. Any change in the vertical dimension will not be permanent if the desirable position of the mandible is not maintained, keeping the opposite teeth apart between meals. On the other hand, if this latter habit is instituted and maintained, we may avoid the necessity of bite raising in many cases.

TRAUMATIC OCCLUSION AND THE TEMPORO-MANDIBULAR JOINT

It has been reported that some cases of deteriorated hearing have been improved by raising the bite. The writer has not had any similar experience. The anatomic relationship of the articulating mandible and the ear is the thin bone lamina between the condylar head and the outer meatus of the ear. It is conceivable that continuous pressure can produce resorption on one side and deposition on the other side of this bone lamella. This may follow when it is realized that part of the intermaxillary pressure is taken up by the periodontal membranes and part transmitted to the temporo-mandibular joint with an accompanying change there.

HISTOLOGIC FINDINGS UNDER FULL DENTURES

Investigations have frequently disclosed areas of necrosis on the borderline of bone and connective tissue under full dentures (9). These areas were found to be more extensive under lower dentures, probably because of the smaller area of support afforded a lower saddle.

Figure 12 is from the ridge of a lower jaw under a full denture, patient 58 years of age. It shows a necrotic strip which has been freed by rear resorption. Outside the resorbed area new bone formation is proceeding in two places, probably a compensatory reaction. It is readily apparent that such a

Fig. 14. Flabby gingiva. (A) fat, (B) fibrous connective tissue, (C) bone. Courtesy: R. Grohs.

transformation in the alveolar bone under a denture makes any accuracy of original impression valueless.

Figure 13 is a lower ridge under a full denture, patient 60 years of age. The extensive necrosis on the border between connective tissue and bone was apparently produced by intense pressure on the denture. Visible are numerous osteoclasts in Howship's lacunae busily removing the bone from the rear. This is the same process of rear resorption with which we are familiar in periodontal sections where the periodontal membrane between bone and tooth become necrotic. After resorption has taken place the bone will have only a remote resemblance to the former outline which was delineated in the original impression. No impression technique can follow such continued metamorphosis.

Figure 14 shows flabby gum which may develop under a full denture almost any place. The ridge shows the contour of the denture saddle with fibrous connective tissue and fat, but no bone. The bone margin is outside the area of the ridge. The denture evidently was kept in the mouth continuously day and night. The constant grinding and pressing caused the resorption of all the bone. Meanwhile the soft tissue kept filling the space provided for it in the denture saddle. Finally the grinding denture moved the gingiva against the bone base and a flabby ridge was the result. Prevention—remove the dentures at night.

The reason given for keeping the dentures in at night is that they sometimes do not fit when first reinserted in the morning. The soft tissue may have shrunk, or swollen. Swelling may be due to irritation by the denture base material, which swelling subsides with rest. But since the disharmony between gingiva and denture on reinsertion is quickly reconciled, this argument is without value.

TRAUMATIC OCCLUSION IN FULL DENTURES

Exacting techniques for full denture impressions have been developed and means evolved for reproducing these in the finished denture. But of what avail are the most

careful techniques if, upon insertion, the patient immediately starts a continuous pressure and grinding, with a resultant continuous resorption of the underlying bone? The response of bone to pressure is resorption.

Occasionally the statement is made that bone deposition may be produced by the exercise of pressure under full dentures. A misunderstanding of the facts is apparent in this reasoning, since a pressure area on bone always leads to resorption. The fact that sometimes a compensating deposition of bone in the area surrounding the resorption develops, (Fig. 12) does not alter the basic tenet.

Whenever possible pressure on bone is translated into traction. This is answered by bone deposition. This is not only the case with muscle insertions but also in the suspension of our teeth. The outstanding exceptions are the bones of the legs when we stand, and the vertebral column. We know how tired we become after a period of standing, and we must lie down in order to rest the vertebrae.

Likewise keeping the dentures in the mouth day and night under almost constant pressure will show its ill effects. The underlying bone is not built to take pressure at all, and in a few months it will bear no resemblance in shape to the contours at the time of the original impression. The degree of pressure varies so that the length of time necessitating a rebase varies in different patients. If the pressure and grinding between meals is eliminated during the day, and the dentures removed at night, the supporting structures are allowed the same rest we allow the long bones of the legs and the vertebrae when we lie down.

Other factors intervene causing the observation that there is a greater tendency in some patients to resorption than others, the pressure being equal. Since it is difficult to prove and we cannot control these factors, we would do well to attend those factors over which we do wield some measure of control.

Dentures should be left out of the mouth at night for ordinary rest and recuperation of the tissues. If the pressure is the cause of necrosis on the border between the connective tissue and the bone, night rest will not suffice to repair such an area. Only an uncomplicated resorption will be relieved by overnight rest.

At times a patient complains of pain under a denture with no obvious cause. In a varying period, the pain may disappear spontaneously. This is probably due to an area of necrosis on the bone surface which is resolved. We have mentioned the same incidence in natural teeth.

RAISING THE BITE IN FULL DENTURES

When rebasing a denture it is often necessary to raise the bite to compensate for alveolar ridge resorption. Lowering of the vertical dimension, often in a very short period of time, should highlight the subject of continuous denture pressure against the bone. To reiterate—short lived pressure generated during meals apparently cannot cause bone resorption. The length of time necessary for osteoclasts to form after pressure stimulation is not precisely known. In dog experiments (7) it was found that not only were osteoclasts present, but some bone resorption had already occurred in 24 hours after stimulation by pressure. We must assume it takes a few hours before osteoclasts appear. And since the chewing process is of short duration, the stimulus will not be sufficient for the purpose. The tendency to osteoclast formation will be removed immediately on finishing with the chewing provided always that we relax immediately and keep the teeth apart. The intention for formation of osteoclasts is dropped before achieving the goal.

Every case presenting an unreasonably short interval for rebasing should immediately pose the question of underlying habits of the patient. The elimination of night grinding with artificial dentures is easy, with the natural dentition it sometimes is a difficult problem. Poor adhesion of full dentures may be a reason for pressure being exerted in the first place.

CONCLUSION

It has been established in the past that the physiologic rest position of the mandible is such that the opposing teeth are out of contact. For some reason, usually nervous people grind and press their teeth continuously. The result in the natural dentition is necrosis of the periodontal membrane; with artificial dentures it produces necrosis of the

connective tissue lining the bone. Both result in considerable bone resorption. Therefore, the physiologic rest position should be taught to all patients, so that they keep their teeth apart between meals. Generally a dorsal position during sleep will take care of the position of the natural dentition. In some cases night splints will be necessary. Dentures should be removed at night to avoid resorption by pressure and to rest the mucous membranes from continuous covering by an impervious material. If we eliminate traumatic occlusion that way it no longer represents a problem. Grinding does not, as a rule, help traumatic occlusion.

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Cancrum Oris*

By R. Gordon Agnew, B.A., M.Sc., D.D.S., PH.D., F.I.C.D.
West China Union University, Chengtu, Szechwan, China

INTRODUCTION

It is fortunate, indeed, that cancrum oris is a relatively rare disease; for those who have had considerable contact with it will agree with me, I am sure, in the opinion that, of all diseases affecting the child, there is, perhaps, no other so frightful in its total picture of devastating facial destruction, extreme rapidity of development and breakdown of body defenses and, until recently at least, very high mortality.

In North America this disease has, for reasons to be discussed, almost become a "clinical curiosity" (8). However, in other less favored parts of the world, this is by no means the case; and, while the incidence of noma in the adult is universally uncommon, the disease occurs in the child over wide geographical areas and to an extent which demands of the health sciences consideration as a public health problem.

Moreover, in addition to its sporadic occurrence, under peacetime conditions in the Western world, the exigencies and frequently appalling environmental conditions attending modern "total" warfare have resulted in a degree of recurrence of this disease, especially in prison and concentration camps affecting both adults and children. In Canada, recently, a periodontist was called upon by governmental health authorities, to offer guidance in the treatment of a member of the armed forces afflicted with this disease known to most health personnel only as a "textbook disease."

This disease, in its wider implications, is integrally related to a most significant aspect of the present world picture, an aspect which demands the attention of all nations. Noma is a strictly preventable disease. Its relative elimination from the public health problems of several Western countries indicates the feasibility of its control. The control of this and of other conditions of fundamentally related origin constitutes an imperative in the achievement of that minimal world health which is a prerequisite to world peace on a reasonably permanent and stable basis. Prevention of noma is not merely a problem of public health instruction and of personal hygiene; it is that and more. It is basically an economic and social problem; it is a part of that major "problem of the underprivileged" which lies at the very core of the present world dilemma.

TERMINOLOGY AND DEFINITION

Noma is commonly defined as "all forms of spontaneous gangrene which involve mucous membranes or muco-cutaneous orifices" (6); "a progressive phagedenic ulcer" (8).

While it occurs most commonly in the mouth, it may occur in other parts of the body, the nose, the eyelids, the auditory canal, the vulva, prepuce, anus. The lesion commencing in the mouth may be termed "cancrum oris" or "gangrenous stomatitis." Terms less commonly employed are "cancer aquaticus" and, because of its high mortality, "malum mortum" (9).

HISTORY

The term "noma" has long been in use. Hippocrates and many subsequent writers have used the term in a general way to indicate destructive and perforating lesions (9); and have not separated this lesion from other mouth and pharyngeal infections (6). It is claimed that Bathus, in the early part of the seventeenth century, first described the symptom complex of noma; and that Van der Voerde made the primary application of the term to this disease picture (9); other sources give to Lund credit for the first use of the term in its present connotation, and refer to the work of Van Swieten in 1728.

Early writers stressed the factor of malnutrition in etiology. This was regarded as the sole cause by some; others noted the recurrent association of noma and exanthematous disease upon a background of malnourishment and debilitation. Henoch used the term "cachexia pauperium" and related the disease to malnutrition and unhygienic

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living conditions. The name "hospital gangrene" derived from its frequent occurrence in the early, unsanitary hospitals.

There is not unanimity regarding contagion. It is frequently described as noncontagious; and attention is drawn to the fact that seldom are multiple cases observed in one family. On the other hand some reports would indicate that the disease may be definitely contagious. Well-known in the literature is the reported instance where, in a hospital, five cases of noma occurred in a single ward; all began in the external auditory canal, in connection with which it was found that one syringe had been used for irrigation in all cases. (We have had no evidence in West China of its having a contagious character.)

DISTRIBUTION

The distribution of noma is such as to include a very large part of the densely populated and moderately populated regions of the world. Cases are reported from China, Japan, India, Africa, Siam, Brazil, Philippine Islands, Malaya, Argentina, France, Germany, Italy, Russia, Turkey, Poland, Spain, United States, England, and other countries.

PREDISPOSING CAUSES

While cancrum oris may occur at any age, it is predominantly a disease of childhood and particularly of the first decade of life, from 2 to 10 or 12 years of age (3) (4) (5) (6), with most cases under 5 years. It affects both sexes, although it would seem to occur more often in females. Reports would suggest that adults are affected especially in the third and fourth decades, although Burkwall (4) is of the opinion that many reported adult cases belong more correctly to that group of diseases based on localized angioneurotic spasm of smaller arteries. In forty cases observed by Eckstein (3) in Ankara, Turkey, during three years spent in a children's hospital, the majority of cases were males.

Data on seasonal incidence in various parts of the world is inadequate. In Turkey the highest occurrence was during the months August to November (the hot season) but in China more cases have been observed during winter months in the Central, West, and North areas (November to February or March). Yet no month was free of cases and the seasonal variation bears an intimate relation to that of malaria.

Jorge and Garcia, quoted by Thoma (10) considered the constitution of the parents as a factor, listing the following conditions: mental disease, tuberculosis, syphilis, rickets, congenital asthenia and alcoholism.

There is widespread agreement among observers that cancrum oris is rarely, if ever, a primary condition. Outstanding among pre-disposing factors is that of malnutrition; and almost all authorities agree that it seldom occurs in a well-nourished healthy child. In West China, where the disease is by no means a rarity, I have never seen a case in which obvious malnutrition did not exist. However, such cases have been reported. Eckstein (3) opposes the claim of avitaminosis as a predisposing cause. The cases in Turkey observed by him occurred during the summer months when, he states, there is ordinarily no shortage of vitamins; he further claims that no symptoms of vitamin deficiency were noted; and he refers to the observation that no two members of the same family with the same (?) nutritional conditions developed the disease. McMillen (1) describes a case observed in Africa as "well-nourished" but gives no detailed nutritional history of any of his cases. Apart from a very few instances, a defective nutritional state would seem to be a *sine qua non* of this disease.

A second basic predisposing factor is generally conceded to be that of unsanitary environment. In Far Eastern countries cancrum oris mainly attacks children of families belonging to the lowest economic and social levels. Unsanitary home conditions and a markedly unhygienic state of the mouth are usually found in conjunction with the malnutrition above described. In addition to the usual problem of the contamination of food, dirty fingers, unclean teeth, the placing of contaminated objects in the mouth, there are such practices as that in which the mother partially masticates food in her own mouth and transfers it to the mouth of the child.

However, since malnutrition is widely prevalent throughout the world, since unsanitary conditions are commonly observed, and since even under these unfavorable circumstances cancrum oris is seldom a sequela, it is obvious that there is a third factor which is usually essential to the development of the lesion.

This third factor is, in most instances, a

state of lowered resistance or debilitation, both local and general. The patient is found to be combating, or convalescing from, one of the acute fevers or from some seriously debilitating disease. Many such conditions have been found: measles, typhoid fever, scarlet fever, diphtheria, chronic malaria, smallpox, kala-azar, tuberculosis, bronchopneumonia, streptococcic septicemia, chronic nephrosis, bacillary dysentery, amebic dysentery, frank starvation, Vincent's angina, during the puerperium, typhus, relapsing fever, pneumonia, erysipelas and enteritis.

Ankara studies (3) indicated the significance of malaria in this connection (29). Eckstein believes that the heavy involvement of the reticulo-endothelial system in chronic malaria is conducive to the loss of immunity which may lead to cancrum oris, whereas he considers measles to be the chief predisposing factor in Western Europe. In 22 cases seen in 1938 he found plasmodia in the blood or spleen of 19 (in 16 cases the parasite was *Plasmodium falciparum;* in 1—*Plasmodium vivax;* in 1—all three plasmodia).

In a study conducted by Zee and Paty in Kansu, China, subtertian malaria complicated 26.6 per cent of 43 cases of cancrum oris (mortality 22.2 per cent of treated cases).

From North-East China observers, including Fan and Scott (25), Struthers (27) and others emphasize the predisposing significance of kala-azar (leishmaniasis), a parasitic disease of the reticulo-endothelial system caused by Leishman-Donovan bodies. This condition is marked by proliferation of endothelial cells which are filled with parasites. In most cases the liver, spleen and bone-marrow are involved. In the proliferation of the enlarged macrophage cells of the reticulo-endothelial system, the lymphatic glands, intestinal mucosa, testes and subcutaneous tissues may also be involved. There is high temperature; rapid pulse; later double-remittent (Pel-Ebstein) type of fever; ascites; edema of the feet; splenomegaly is a marked feature; anemia; thrombocytopenia; granulopenia; hemorrhages from nose and gingivae. Noma may begin as a small ulcer on the palate. From 5 to 15 per cent of kala-azar cases studied by Struthers (27) and Chu and Fan (11) developed noma; and 95 per cent of noma cases studied in Honan province (27) were related to kala-azar. Fan and Scott (25) reported 6 cases of noma in 261 of kala-azar; and of 57 cases of noma, 40 were related to kala-azar. (Above references are to visceral leishmaniasis in contradistinction to cutaneous leishmaniasis or "oriental sore" in which characteristic lesions may occur on the face or elsewhere.)

Numerous observers have referred to the importance of the blood dyscrasias in the development of cancrum oris. Giraud *et al* (7) describe a case of leukanemia of lymphoid type with terminal noma due, in their opinion, to the extreme debilitation and lowered resistance of the patient. Seifert (5) reports three cases (2 of typical agranulocytosis and 1 of leukemia) and states that "myelophthisis is the cause of noma as well as of other related diseases." Thoma (10) suggests that blood dyscrasias (i.e. in a casual relationship) are probably more common than supposed. A marked neutropenia has been noted immediately prior to occurrence of cancrum oris. Hicken *et al* (8) describe a case of acute myelogenous leukemia in a child of less than 4 years (an unusual occurrence at that age), complicated by noma and by general acute streptococcic dermatitis ("hospital pemphigus") —a trio of uncommon findings. Histopathologic study disclosed no evidence of leukemic change in the noma ulcer. Their bacteriologic studies convinced them that noma is not caused by leukemic infiltration of the skin and mucous membranes, but is an infective gangrene (see below); that it is not part of the leukemic process. In fact, since, in the case described, the noma process showed evidence of cessation before the patient died, and since the blood picture materially improved following the favorable development in the mouth, they suggest that the blood picture of leukemia is an abnormal response to—in this instance—the noma infection stimulus. At the Peking Union Medical College it was noted that when noma developed there was a coincident drop in granulocytes.

Obviously, most instances of blood dyscrasia are not complicated by a true cancrum oris; and, historically, many cases of cancrum oris have no such blood picture, so that evidence is lacking for the establishment of an intimate relationship of common occurrence. Convincing evidence of the causal role of the mouth lesion in the

development of some blood dyscrasias is similarly lacking. Apart from the possibility of mere coincidence, however, the view would seem tenable that the blood dyscrasias constitute factors potentially leading to the state of lowered resistance which is seemingly necessary for the development of the noma lesion.

Trauma and its sequelae would seem, in many instances to stand in a causal relation. Many reports indicate the relation between the extraction of teeth and the subsequent determination of the point of commencement of the lesion. In this regard, the extraction of partly erupted third molars is emphasized (10).

Accidental biting of the cheek is another form of trauma which has led to the development of cancrum oris. Hicken *et al* describe a case in which a young child, while chewing gum, bit the inner surface of the right cheek. Within two days the entire cheek was swollen and painful; and in four days a large typical sloughing ulcer had involved the inner surface of the cheek, with typical foul odor, and other symptoms; in spite of local treatment it continued to extend. Davis (13) describes the case of an American seaman, aged 21, stationed at Cheefoo, China, who similarly bit his cheek while eating. Within five days a typical cancrum oris lesion had developed.

From Japan, Ishikawa (16) has described a case of psoriasis vulgaris upon which cancrum oris developed six weeks after admission to hospital. The patient was a man, aged 56. In the opinion of the clinicians the very severe generalized psoriasis constituted the background for the appearance of the noma lesion. They observe that, under Cignolin treatment the psoriasis condition was showing marked improvement; but the mouth lesion and related heart involvement led to the death of the patient.

Cancrum oris occurs frequently in India. In one instance Gokhale (14) describes the case of a woman, aged 30, contracting the disease following puerperium, in spite of vigorous treatment. It was considered that hemorrhage and sapremia during the puerperium constituted the resistance-lowering factor.

Very unusual is the case described by Diamond (9) in which noma complicates a non-specific ulcerative colitis. He considered that the marked general toxemia accompanying this fulminant type of ulcerative colitis must be viewed as the predisposing factor. The patient did not recover.

From Africa, McMillen (1) describes noma as a complication with yaws and bronchopneumonia. (Yaws is caused by the *Spirochaeta pertenuis)*; this unfavorable background slowed down treatment; and the yaws lesion furnished a rich soil for the spread of the noma lesion.

Hitzelberger (21) describes a case of an American police officer who developed a fatal cancrum oris of the fulminating type. He had been a public blood donor, and had given 72 transfusions over a five-year period. Myelogenous leukemia was involved in the disease picture.

References are made in the literature with relation to the possibility of an association between cancrum oris lesions and malignant growth development. Definite evidence in this connection is not available.

CLASSIFICATION

Due to the difficulty of securing adequate material for study and analysis, few attempts have been made to evolve a classification of the various forms of cancrum oris appropriate to the collective clinical experience of this disease. Many writers refer to marked difference in degree of severity; to those cases in which little or no capacity of the tissues for defensive response is evidenced; to those in which a relatively vigorous response may lead, successfully or otherwise, to an attempt at demarcation of the gangrenous zone and delimitation of the phagedenic process. Many points of origin of the necrotic process are described, around teeth, gingival areas or buccal or labial zones, but no comprehensive classification has, to date, appeared. Hill (18) refers to two types: 1. A definitely circumscribed ulcerative process limited in its scope by active granulations in the adjacent tissues. This he terms "gangrenous stomatitis." 2. A rapidly progressive gangrene, involving large areas of the oral tissues, against which there is little evidence of protective reaction. This he calls "True Noma." Various general classifications have been made—for example, according to duration as acute, subacute and chronic; according to degree as mild or severe.

Kohn (19), of the dental staff of the

West China Union University College of Medicine and Dentistry, proposes the following classification:

I. Gangreno-stomatitis buccalis:

A. *Mitis*—This condition usually occurs in children above 1½ years of age. The swelling of the face shows a tendency towards spreading into the soft tissues. Extraorally, marked cellulitis can be seen; while intraorally there is a large necrosed ulcer on the inner wall of the cheek which tends to spread toward the gingivae and also in an anterior direction. Periostitis of the maxilla and later necrosis of the bone are observed. (This condition has been noted every year in West China, following light measles or dysentery.) A late complication, arising perhaps months or years later, is scar formation in the buccinator muscle which adheres to the gingivae, thus causing complete trismus. There is no great tendency towards perforation of the cheek. Prognosis is good.

B. *Gravis*—The mitis type, if neglected, or if the patient's general condition is poor may progress to the gravis type, which is characterized by great destruction of the alveolar bone, maxilla, and adjacent soft tissues of the cheek and face. However, the gravis type may commence as such (as for example, in West China, following the smallpox epidemic of 1946, a gangrenous stomatitis developed with very rapid spread and destructive tendency). This type is called gravis because of the great destruction involved and not because of its endangering the patient's life. It occurs mostly in children over two years of age and runs a very chronic course. The patients are not very toxic.

II. Gangreno-stomatitis labio-mentalis

This type is seen, for example, in mild cases of diarrhea, especially in the autumn (in West China). The entire mouth presents an appearance of general necrotic (Vincent's) gingivitis; but, on closer examination, a deep necrosed ulcer with a gangrenous slough may be seen localized in the fold between the gingivae and the labial mucosa. The condition tends to be central in location, and there is no great tendency towards perforation of the soft tissues of the chin. In neglected cases necrosis of the mandible develops; this heals in a manner comparable to that of chronic osteomyelitis. Prognosis is good.

III. Gangreno-stomatitis labio-maxillaris

The most severe cases fall into this category. The lesion occurs chiefly in very young children. There is usually no swelling of the surrounding tissues, nor hyperemia around the lesion. The location is in the upper lip, around or near one corner of the mouth, with a typical darkish area of limited size seen extraorally, and with a necrosed ulcer in the same corresponding location intraorally. In a very early stage there is no lesion intraorally. This type usually progresses to perforation very rapidly, destroying the underlying bone in various degrees. (In West China it is seen mostly in cases of severe measles or dysentery; it was very prevalent in Chengtu following the 1943 epidemic of measles.) There is a tendency to spread to the nose and canine fossa, with destruction of adjacent tissues. The patients are completely apathetic and very toxic. Prognosis is very bad.

GENERAL CLINICAL PICTURE

The classical picture of the symptoms and lesions of cancrum oris has appeared frequently in the literature and is, on the whole, dependable. However, as indicated above, paucity of clinical material has resulted in frequent failure to recognize with clarity the variant types with variant degrees of severity and variant prognoses. Thus, for example, based upon the few cases ordinarily available, emphasis has been often laid upon the absence of protective inflammatory reaction; or, conversely, upon the tendency toward delimitation and demarcation; or, upon a specific linkage with one of the many possible background factors which may play an essential role in the inauguration of the disease. An obvious difficulty is that, since the initial lesion occurs, usually, within the mouth, the mother of the child may fail to discover its existence; progress may be so extremely rapid that, by the time it is discovered and the little patient taken to a dentist or physician, the earliest lesion is masked by more advanced stages of development. Usually the parent or the child itself is able to describe the early changes with only very approximate clarity or accuracy.

It is very possible that early descriptions may have, to some extent, associated ulcerative stomatitis with noma; or mercurial stomatitis with noma. Many have seen ulcerative stomatitis develop into cancrum oris.

The characteristic foul odor may often constitute the first extra-oral evidence of the disease. Examination of the mouth may show a painful red or purplish-red spot or indurated papule on the buccal surface of the cheek, the labio-gingival fold or about the lips (*See* classification). This rapidly breaks down to form an ulcer. When on the buccal surface this may be in contact with a similar lesion on the alveolar mucosa (18). In the relatively benign forms the ulcer may be deep but may be well circumscribed with evidence of granulation tissue formation. Marked cellulitis may be present.

In the severer forms little evidence of defense may be seen. The initial areas may become confluent and break down into rapidly progressive ulcers. The tissues of the cheek become usually greatly edematous and swollen to a brawny hardening; the skin is stretched and shiny. Shortly there appears a bluish discoloured area on the external surface of the cheek opposite to the original intra-oral lesion; this in turn breaks down to form a perforating ulcer which destroys tissue indiscriminately and with astonishing speed. The affected side of the face becomes completely involved and the eye becomes closed. Within the mouth, following destruction of the soft tissues the alveolar and maxillary or mandibular bone are attacked; the teeth and bony structure become exposed and later the teeth exfoliate; large sequestra of bone may form.

The lesion is usually unilateral but, by extension, may become bilateral. In most instances the cervical lymph nodes become swollen, usually discrete, hard and painful; this varies with the severity of the condition. The necrotic tissue turns black. The extremely foul odor which penetrates to a considerable distance, and the marked destruction of tissue combine to produce a picture of offensive mutilation equalled probably by no other disease. Secondary infection rapidly occurs.

The initial lesion may be located in proximity to the teeth, and usually related to broken-down and irritating hard tissue or to periodontal lesions.

In adults, in China, the disease commonly follows a less severe course than in children. Recovery may occur spontaneously, possibly after extensive disfigurement. Burket (20) points out that in America in edentulous patients, the gums or alveolar ridges may be first involved; and that in aged individuals an ischemic dry gangrenous process usually precedes the more infectious state, suggesting a close relation between vascular disease and the oral gangrene. Often there is some chronic disease that affects directly or indirectly the cardio-vascular system, (ex. chronic glomerulonephritis).

A characteristic of the disease in children is the steep increase in salivary secretion. It is felt that this copious salivary flow may exert a definitely deleterious effect in accelerating the spread of the disease process.

Pain is a variable factor in the clinical picture. In most instances the relative absence of pain is a noteworthy feature. In a minority of instances pain is present, although rarely to a marked extent. Apathy is usually evident, especially in the malignant types; general prostration and drowsiness, weak pulse, muscular relaxation, and, sometimes, diarrhea.

The temperature may vary within wide limits. Frequently it is elevated to 102° or 103°, or higher; often hectic in type. Pulse and respiration may be markedly increased. Delirium may be, in rare instances, associated with high fever. Rise of temperature may indicate a general septicemia. Noma may progress without fever, even with extensive facial destruction (3).

Noteworthy in the blood picture is the frequent absence of a vigorous leukocytic response; this condition is reflected in the histological section suggesting (3) that, as necrobiosis progresses in noma, there is no remarkable response on the part of the leukocytic system. Along with a commonly low white blood cell count there is usually a definite anemia. The possibility, as referred to under "Predisposing Causes," that the noma stimulus may lead to certain disturbances of the hematopoietic system should be kept in mind.

BACTERIOLOGY

Bacteriological studies of the noma lesion have yielded a large number of different organisms. From the standpoint of ultimate proof no single specific organism or no synergistic grouping of organisms has yet been established as the immediate etiologic agent. However, there is almost universal agreement in allocating significance to the presence, in symbiotic relationship, of the *Bacillus fusiformis* and the *Borrelia vincenti*. The

view is widely held that, from clinical, bacteriological and histological standpoints, ulcerative stomatitis and noma are practically identical, the noma constituting a more advanced or more toxic form of ulcerative stomatitis. In other words, noma would take its place as one of the forms of fuso-spirochetal disease individualized only through its localization and its rapid and unlimited development. Lingard in 1883 was the first to observe these organisms in smears from cancrum oris.

However, many other organisms have also been found. These include: hemolytic streptococci; non-hemolytic streptococci; diphtheroid bacilli; gram-positive and gram-negative cocci; bacillus coli; *Bacillus paracoli idolicum; Bacillus mycoides; Bacillus subtilis; Bacillus proteus; Micrococcus tetragenus*; gram-negative bacilli (possibly the anerobic *Bacillus ramosus*); Bacillus of malignant edema, etc.; leptothrix and cladothrix forms; etc.

Hicken *et al* (8) report the following findings:

a) *From sloughing gangrenous area*—1) *B. fusiformis;* 2) *Borr. vincenti;* 3) Non-hemolytic streptococci; 4) *Staphylococcus aureus;* 5) Diphtheroid bacilli; 6) Gram-positive bacilli.

b) *From zone of reaction*—(i.e. junction of inflammatory and gangrenous areas) 1) Anerobic *B. fusiformis;* 2) anerobic microaerophilcic non-hemolytic streptococci; 3) anerobic staphylococci.

Cultures of the above combined organisms emitted a foul odor seemingly identical with that of clinical cancrum oris. These authors have concluded that the noma process requires the symbiotic relation, therefore, of 1) non-hemolytic streptococci; 2) *B. fusiformis;* 3) stapnylococci. To establish this, they point out that when rabbits were injected with a non-hemolytic streptococcus, an acute localized dermatitis resulted; but that all lesions healed within one week. When bacterial suspensions of *B. fusiformis, Staphylococcus aureus* and non-hemolytic streptococcus were injected, a large, sloughing, gangrenous ulcer of skin and subcutaneous tissues occurred. On the other hand many investigators have described, in the innermost zone of tissue an invasion into apparently healthy tissues of the *Borr. vincenti.* Eckstein (3), using a new method of pricking healthy material just beyond the necrotic portion obtained fluid with a capillary tube; this fluid microscopically showed *B. fusiformis; Borr. vincenti;* a few leucocytes, but no other organisms.

In advanced cases, superficial cultures of the lesion may show non-hemolytic streptococcus but no *B. fusiformis* or *Borr. vincenti.* These latter organisms may be found in the deeper strata after removal of the slough. According to many, the *Borr. vincenti* is the essential cause of the necrosis; it forces its way into the healthy tissue "as though paving the way for the *B. fusiformis*"; complete necrosis can only occur if the *B. fusiformis* is able to penetrate the tissue in company with the Borrelia. Vincent observed that, when *B. fusiformis* was found alone, the process was milder without deep ulceration; the *B. fusiformis* causes inflammation and ulceration, whereas the brownish-green or black gangrene is due to various bacilli and cocci growing on the surface. Absence of the symbiotic organisms in healthy tissue beyond the necrosis could be interpreted as a favorable sign.

PATHOLOGY

Microscopically, three zones may be distinguished: a) *Gangrenous area:*—In this extensive zone all cells have lost their morphological characters; nuclei are pyknotic and fragmented; cell walls have disintegrated; tissue stains more or less homogeneously *en masse* with fibrin and red cells. Masses of organisms of various types are present (6) (8) (17). b) *Intermediate area or Zone of reaction:*—Here is seen a slight degree of infiltration, with polymorphonuclear leucocytes; some growing connective tissue cells are present; the walls of the blood vessels are slightly thickened, their lumina filled with obstructing thrombi. *B. fusiformis* and *Borr. vincenti* are numerous and other organisms may be present; they may invade the vessels, the walls of which early show coagulation necrosis. Most noteworthy is the minimal degree of cellular reaction.

c) *Healthy Zone:*—The transition from zone two to three may be remarkably abrupt due to the already noted absence of appreciable tissue reaction. As the normal area is approached, vessel lumina are found to be patent but may contain many disintegrating RBC. In this inner zone considerable numbers of *Borr. vincenti* may be found, seem-

ingly arousing no response on the part of the tissue.

In the benign forms of the disease, where recovery may occur, there may be considerable cellular response in the form of a barrier, lymphocytes, polymorphonuclears, monocytes, etc., more or less effectively walling off the necrotic area.

In some instances only two areas, the necrotic and healthy zones may be differentiated, the line of demarcation sharply defined; on the necrotic side the tissue completely disorganized and the vessels thrombosed. In the immediately adjacent healthy area there is no thrombosis or hyperemia of the vessels. *Borr. vincenti,* and in some cases *B. fusiformis* are frequently seen invading the otherwise healthy tissue.

It has been suggested that the gangrene may be due to possible blocking of the circulation with Vincent's organisms (Blair and Ivy).

EXPERIMENTAL STUDIES

Gangrenous attack of tongue and cheeks and secondarily of lungs has been reported by Lingard in 1888 as affecting not only man but monkeys, calves and pigs. He claims to have produced noma in calves by the inoculation of gangrenous material from the horse lung. Smith (6) questions the true nomatous nature of these lesions and the possibility of the involvement of the *B. necrosis* instead. His own experiments, using well nourished guinea pigs inoculated with fuso-spirochetal material did not produce the typical noma lesion. Subsequent controlled experiments using vitamin-C deficient guinea pigs, yielded suggestive results. Fuso-spirochetal material from mouth of a patient with periodontal disease was inoculated into the cheeks of the pigs between the mucous and the cutaneous layers. The well nourished controls mostly developed local abscesses but recovered. Of the 10 vitamin-C deficient animals, three died in 48 hours without obvious lesions; the other seven developed black gangrenous lesions of the cheek similar to noma, and died in 4 to 8 days. This would seem to indicate experimentally the background role of dietary deficiency.

Experiments in Vitamin B-2 complex (21) (22) (23) (24) in monkeys resulted in production of lesions clinically resembling noma.

In India cancrum oris developed in a monkey which had been experimentally infected with *Leishmania donovani* four months earlier.

Reference has already been made to experiments on the rabbit with non-hemolytic streptococcus, *B. fusiformis* and *Staphylococcus aureus.*

PROGNOSIS

It is not possible to indicate in other than very general terms the mortality of cancrum oris. This stems largely from the over-all paucity of material and the absence, in most reports, of a differentiation between mitis and gravis forms. Most general statements indicate a mortality, in untreated cases, of from 75 to 95 per cent. The group of cases in which defensive reactions take place would obviously show a much lower lethality. The North China type of noma complicating kala-azar reacts favorably to treatment. Struthers (27) estimates a mortality of 75 per cent in untreated cases in Honan Province, dropping to 5 to 10 per cent in treated cases. Fan and Scott report (25) 85 per cent success in treatment in Shantung Province. Chu and Fan (11) report an over-all mortality of 55 per cent in Peiping, studying 100 cases, 79 of which were related to kala-azar. In West China, where we have had contact with this disease over many years, success in treatment, always gratifying in cases of early presentation, has been greatly aided since the introduction of the sulpha drugs.

In view of the phagedenic nature of the lesion there are few diseases in which promptness of diagnosis and treatment, after first evidence of nomatous change, affects so profoundly the outcome.

Future mortality rates will be determined by many factors: 1) the efficacy of new therapeutic agents—the sulpha drugs; penicillin and related forms; 2) effectiveness of national and international mechanisms of food distribution and economic organization; 3) public oral health education.

It would seem that noma leaves no local immunity after healing (3). Cases have been observed in which noma developed for the second time and gradually advanced on the site of the old, and presumably, healed lesion; this recurrence may follow after weeks or months and may be related to injury, traumatic irritation, plastic opera-

tions, and subsequent lowered resistance. If any local immunity is developed, it would seem to be of short duration.

Death occurs from secondary infection—from septicemia; from pulmonary gangrene as terminal complication, probably due to aspiration of necrotic material from the mouth. A serious complication may be an intractable diarrhea presumably caused by the swallowing of necrotic oral material, or by a bacillary dysentery. Broncho-pneumonia is one of the commonest immediate causes of death in kala-azar and other cases (27).

Related to the finding that oral hygienic conditions constitute one of the usually basic background factors is the fact that mortality in noma is definitely lowered in cases where adequate oral cleanliness can be maintained.

DIAGNOSIS

Diagnosis of cancrum oris offers usually little difficulty, as indicated in connection with the general clinical picture. Mead (17) states, with brief accuracy, that there is nothing else like it. The differential diagnosis should take into account other ulcerative conditions such as agranulocytic angina (15). However, this condition shows frequent hemorrhage and is less extensive; there is usually no hemorrhage in noma. The blood picture usually indicates anemia; and may resemble agranulocytic angina and the leukemias; or may indicate only a low leucocyte level.

In the tropics the so-called oral lesions of leishmaniasis (27) are doubtless often true noma of the type already described in connection with kala-azar (i.e. the visceral leishmaniasis or kala-azar). The lesions of cutaneous leishmaniasis (Oriental Sore) may offer a differential diagnosis problem, but this is unlikely.

Manifestations of syphilis, yaws, gangosa, etc., must be kept in mind; also postoperative surgical ulceration (possibly due to amoebic contamination (15); also inguinal venereal ulcers; and certain malignant growths (ex. malignant granulomas) and underlying carcinoma in adults (6). Lesions of gas gangrene due to spore-bearing anaerobes, and lupus vulgaris might on occasion require differentiation (6) (32).

Bacteriological examination will seldom be required for diagnosis. When carried out caution must be used in interpretation of the presence, absence, and quantitative presence of the fuso-spirochetal organisms, as outlined under "Bacteriology". Similar care is needed in biopsy examination, in which study the presence of substantial tissue response and absence or relative absence of *Borr. vincenti* in healthy tissue may be interpreted as favorable in prognosis.

AUTOPSY FINDINGS

Reference has already been made to histopathological observation of the local lesion, as seen in postmortem study or in biopsy. Noteworthy is the slight evidence of tissue reaction; such infiltration as exists with edema may be of round-cell or polymorphonuclear type. Masses of *B. fusiformis* seen in and on superficial areas of necrosis; invading spirochetes, often with *B. fusiformis* seen in surrounding healthy tissues.

General autopsy findings vary widely, in view of the many pathological states associated with the noma process.

TREATMENT

Treatment of cancrum oris varies within wide limits in different areas. Lack of a recognized specific causative factor has naturally resulted in considerable divergence in therapy. However, certain fundamental conditions are generally accepted. Early diagnosis and prompt treatment are of the highest importance. While some emphasize general treatment to the relative exclusion of local, and while others follow the exact converse—there is general unanimity in emphasis upon the drastic improvement of the nutritional state of the patient. Massive doses in multi-vitamin therapy, esp. A; B complex; C and D; high caloric diet; high vitamin-content, and high-protein foods. If lesion affects lips and gingivae, food should be administered by nasal intubation, through tube no larger than size 10 catheter into stomach; care is taken not to injure adjacent normal appearing tissues. Patient is hospitalized and kept warm and quiet. Blood transfusions should be given if and when necessary.

Burkwall (4) lists 18 drugs or therapeutic procedures which have been suggested or employed. Since appearance of his publication, additional drugs or procedures have been used. A list would include—1) surgical excision and cautery of wound edges

with either chemical, thermal or electric cautery; 2) escharotics; 3) hot fomentations; 4) hydrogen peroxide; 5) mild antiseptic dressings; 6) 10 per cent formalin; 7) formaldehyde undiluted; 8) typhoid vaccine; 9) arsphenamines, both locally and intravenously; 10) deep X-ray therapy; 11) tartar emetic (esp. for kala-azar); 12) emetine; 13) methylene blue crystals; 14) zinc chloride mouthwash (dil. 1:2000, every hour); 15) 5 per cent chromic acid painted 3 times daily; 16) phenol; [formula: iodine —4 parts; phenol—5 parts; alcohol—1 part]; 17) local oxygen gas instillation; 18) hypertonic saline solution intravenously; 19) blood transfusion; 20) sulphanilamide; 21) anti-diphtheritic serum; 22) antigangrene serum; 23) ultra-violet radiation; 24) infrared radiation; 25) metaphen; 26) potassium permanganate; 27) sodium perborate; 28) copper sulphate; 29) iodoform; 30) gentian violet; 31) acriflavine; 32) penicillin.

Following general measures, the institution of appropriate dietary regimen, etc., prompt surgical excision, perhaps the oldest form of treatment, is still of high importance in most cases. Resection may usually be done (and best done, if possible) without any anesthesia; gangrenous tissue is removed and excision is carried well into healthy tissue, loose teeth are removed; root cavities curetted; usual surgical precautions and after-treatment. Use of electro-cautery obviously most appropriate. Hesitancy in free excision, on grounds of further mutilation of face, should not be permitted, because of grave danger of extension of the disease process; plastic repair may be instituted later.

Asbell (2) claims prompt elevation of leucocyte count upon administration of sulphanilamide; also subsidence of toxic symptoms and delimitation of gangrenous area. (His case was doing well when separating slough caused carotid branch to rupture and patient died.)

McMillen (1) treated seven cases with complete success (previous mortality rate had been 90 per cent) in Africa with formaldehyde solution. He claims that the arrest of noma may be definitely delayed by saliva or tears; hence the dehydrating action of the undiluted solution of formaldehyde seems important. The odor of formaldehyde is noticeable 24 hours after the solution has been applied; and possibly the action of this gas, given off over a long period, is of some importance. Contrary to the principle, above outlined, of excising freely and into healthy tissue, McMillen feels that it is important not to excise all of the necrotic tissue, because such tissue is spongy and tends to hold much more of the formaldehyde solution and to give off gas for a much longer time than does healthy tissue.

To protect the lungs against the irritating vapor of formaldehyde McMillen places cotton in the back of the mouth while applying the solution; and into each nostril if swabbing the nasal area. The ulcer is dried with cotton before application of the chemical; and applications are made once daily until no sign of further extension and the ulcer has become dry and free from stench; four or five treatments are usually necessary.

Eckstein (3) reports 8 failures out of 9 with neoarsphenamine (1 early case recovered). However, using antigangrene serum he was successful in 11 out of 21 advanced cases. He claims that the beneficial effects of this method lie in the non-specific action. Since the tissue affected by noma is often not sharply demarcated from the surrounding healthy tissue and since an inflammatory reaction in this zone is a good sign that there is an inclination toward healing, he injected serum locally in the healthy tissue adjacent to the necrotic area, using 100 cc. serum; also used 10 to 20 cc. serum intramuscularly and an equal amount intravenously; this treatment was repeated daily until sloughing of the necrosed tissue took place (average total of circa 500 cc. serum used).

In West China we have had excellent results following prompt and vigorous nutritional fortification (using locally available foods, with concentrated vitamins, and fortified milk powder) and free surgical excision. Adjunct treatment of various types alternated, and was directed toward the elimination of the *B. fusiformis* and *Borr. vincenti.* Sulpha drugs have proved of distinct value in recent noma therapy in this region (Note Chart, from records of the West China Union University Dental Clinic, supplied by A. W. Lindsay. In the small series here listed, healing resulted in approximately 80 per cent of the cases).

Penicillin would seem to be a valuable adjuvant and to offer therapeutic possibili-

RESULTS OF TREATMENT OF NOMA WITH SULFA DRUGS

(11 Advanced cases treated from April 1945 to Aug. 1946)

West China Union University Dental Clinic

Case	Sex	Age	Drug	Dosage			Effect on Lesion		Result
				Daily	Duration	Total	Controlled	Separated	
1	F	6	ST	3.0 gm.	4 days	12 gm.	5th day	5th day	healed
2	F	2	ST	1.5 gm.	3 days	4.5 gm.	4th day	4th day	healed
3	M	3	ST	2.0 gm.	4 days	8.0 gm.	4th day	5th day	healed
4	M	1½	ST	1.5 gm.	2 days		(did not	come again)	
5	M	3	ST	2.0 gm.	2 days		excision		
			SM	1.5 gm.	1 day		on		
				0.9 gm.	1 day	6.4 gm.	2nd day	3rd day	healed
6	M	5	SM	2.0 gm.	4 days	8.0 gm.	4th day	5th day	healed
7	M	5	SD	*2.0 gm.	2 days				
				4.0 gm.	3 days	18 gm.		5th day	healed
8	F	2½	SM	3.0 gm.	2 days	6.0 gm.	3rd day	3rd day	healed
9	F	2	SD	†2.0 gm.	1 day				
				3.0 gm.	2 days	8.0 gm.	5th day	5th day	healed
10	F	2½	SD	2 gm.	4 days	12 gm.	3rd day	3rd day	healed
11	M	2	SD	1.5 gm.	2 days				
				2.0 gm.	1 day	5.0 gm.	3rd day	4th day	healed

*Patient took 2 gm. only of drug although 4.0 gm. prescribed.

†Patient did not return following the first treatment until the 4th day. Data supplied by Dental Clinic per A. W. Lindsay.

ties of distinct promise. Experience in the treatment of noma with this agent is still limited and its full value yet to be determined (28). Dawson (26) combines penicillin and sulphanilamide. He has insufflated penicillin-sulphanilamide powder (5000 units per gram) twice daily (or has given parenterally) with good results. After 48 hours the ulcers are clean, (although the patient may die from the general effects of the disease). He does not clearly distinguish, in his report, between mitis and gravis forms. The prognosis, with penicillin, would seem to be good if the lesion is slight or if the mucoperiosteum is intact; if the mucoperiosteum has sloughed from the bone, slow necrosis is inevitable and cure greatly delayed. He continues the penicillin-sulphanilamide powder twice daily until the stitches of the preliminary repair operation are removed; and defers plastic surgery or prosthesis until the patient's health has cleared.

Intercurrent treatment for background conditions present, ex. malaria, etc., is not discussed (for ex. the use of neostam, neostibasan, solustibasan and ureastibamine in treatment of kala-azar).

PROPHYLACTIC TREATMENT

Since there would appear to be a definite relationship between fuso-spirochetal organisms found in the supposedly normal mouth, in fuso-spirillary gingivitis and stomatitis, in Vincent's angina and in noma—depending upon the local and general resistance of the patient and the virulence of the organisms, preventive measures related to noma would obviously be directed toward the early control of the less severe conditions listed, the maintenance of a healthy mouth state, and the establishment of genreal high resistance.

Stagnation areas must be eliminated. In concentration camp studies (26) no case passed on from "Trench mouth" to noma after treatment was commenced. Where the lesion had progressed to noma, stagnation areas were always present (*Cf.* "incubation zones" of Box (31)).

SUMMARY

1. An outline of present knowledge with regard to cancrum oris or noma is presented.

2. Findings in studies of this disease in China are discussed.

3. A classification, possibly the first detailed attempt in this field, by a member of the West China Union University staff, is presented.

4. The feasibility of control of this heretofore highly lethal disease is discussed and emphasized.

5. The wider implications of its control and elimination from human society are presented as a problem and obligation of the healing professions, integral with the strictly professional and scientific consideration of the disease.

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Periodontosis in the Spider Monkey— A Preliminary Report

By HENRY M. GOLDMAN, D.M.D., *Boston, Massachusetts*

INTRODUCTION

Periodontosis may be defined as a degenerative non-inflammatory disease affecting the supporting structures of the teeth. This condition was first described by Gottlieb in 1920, who termed it diffuse atrophy of bone; later he amended his original observations, stating that the disease should be called cementopathy. Box (1924) also described a type of periodontal disease which clinically substantiated Gottlieb's earlier description. Since that time several investigators have discussed the histopathologic changes of periodontosis, among which were Gottlieb and Orban (1933), Thoma and Goldman (1937), and Orban and Weinmann (1942).

The purpose of this preliminary report is to describe the histopathologic changes in the jaws of two spider monkeys affected with a similar condition to that of human periodontosis. It is also intended to correlate these microscopic findings to the clinical manifestations observed in these monkeys.

MATERIAL

This study was made on the jaws of two spider monkeys (Ateles Geoffroyi) which originally belonged to a group of twenty-five animals used for behavior and nerve regeneration experiments. These monkeys were kept in a large enclosure and were fed exclusively on waste, fruits, and vegetables. During the experiment the monkeys became infected with amebic and bacillary dysentery. Six died and in all cases showed ulcers of the stomach or intestine, in most cases with perforations. The two jaws studied came from the last group mentioned. The jaws were examined clinically, radiographically, and microscopically, the clinical observations being augmented with information of the monkeys while they were alive.

CLINICAL FINDINGS

One of the initial symptoms noticed was migration of the teeth, manifesting itself by either extrusion or lateral movement; another was loosening of the teeth. Although no gross distinct inflammatory process of the gingivae was noted, there was a great deal of bleeding. The animals had much difficulty in mastication.

Clinical examination of the jaws showed the gingivae rather pink and smooth, no hyperplasia being noted; however, all degrees of pocket formation were found. Both firm and loose teeth were encountered, some almost being exfoliated. In no single instance was there a pocket present without some loosening of the tooth. Where pockets were present, a slight retraction and rolled margin of the gingiva could be detected; there was for the most part no obvious recession. Calculus formation was abundant, being of the softer type; however, calculus was not found associated with any of the pockets.

X-ray examination disclosed similar characteristic findings to that of human periodontosis. Widening of the periodontal space varied, some teeth being markedly affected while others were not. The widening could be found localized to one surface of the tooth or encircling the entire tooth root; in some instances it was seen in the marginal periodontal area showing up as a cone-like radiolucent area. Figure 1 shows the radiogram of a hemisection of a maxilla. Widening of the periodontal membrane can be seen in the marginal area of the first premolar and around the mesio-buccal root of the first molar, Figure 2, the radiogram of a hemisection of a mandible, shows a marked width of the periodontal membrane around the last molar and on the distal aspect of the second premolar. Sockets of two molars recently exfoliated can be seen. These radiograms represent typical findings.

HISTOPATHOLOGY

Changes in the periodontal membrane. The periodontal membrane showed the main changes of this disease. The early alterations appeared as a loss of the functional structure of the periodontal membrane, the principal fiber apparatus becoming disorganized. The fiber bundles which under normal conditions are attached from the cementum of the tooth to the bone, appeared broken, under-

going degeneration. There was loosening and swelling of the interfibrillar substance and disintegration of the fibers, finally becoming a loose edematous connective tissue. An increased number of capillaries and edema of the tissue were constant findings. The change could be found localized in relatively small areas of a periodontal membrane and in other instances affecting the entire structure.

This disintegration of the principal fibers seemed to arise in the middle portion of the fiber, the attachment to the cementum and bone persisting for a time (Fig. 4). Even in the late stages of the destruction were old fiber attachments found in the cementum. These connective tissue changes were of a degenerative character and were not associated with any inflammatory reaction in the periodontal membrane.

Figure 3 shows a high power photomicrograph of the principal fiber apparatus showing the early stages of degeneration, (Wilder's silver stain to show up the connective tissue fibers). The fibers are swollen and nuclei of the cells are somewhat pyknotic. The blotchy nature of the fiber is evident. Sharpey's fibers are attached to the cementum and bone.

A later stage of destruction is seen in Figure 4. Sharpey's fiber attachments to cementum and bone, however, are still evident. Increased edema and numerous capillaries are seen. No resorption of the bone can be detected. In Figure 5 the entire principal fiber apparatus has disappeared and only but few, if any at all, of the Sharpey fibers can be found. The periodontal membrane now consists entirely of loose, hyperemic, edematous connective tissue. This is the end result of degeneration of the periodontal membrane. One may notice that no bone resorption can be detected.

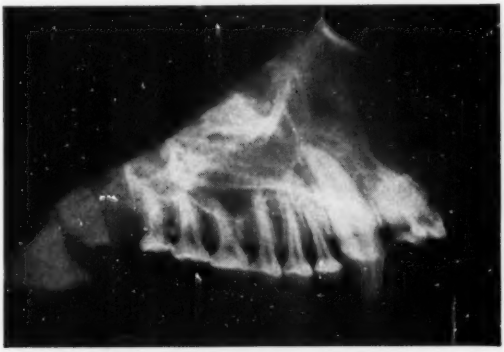

Fig. 1. This is a radiogram of a hemisection of a maxilla. Note the widening of the periodontal membrane in the distal marginal area around the first premolar and encircling the mesiobuccal root of the first molar. These are early radiographic changes.

Fig. 2. This is a radiogram of a hemisection of a mandible and shows widening of the periodontal membrane around the last molar and on the distal aspect of the second premolar. Sockets of recently lost teeth are evident.

Migration of the teeth was a constant finding and was due to the destruction of the periodontal membrane, causing a pull to be exerted by a remaining intact attachment of the other side of the tooth. This movement was also aided by the increased pressure of the edema and proliferation of capillaries on the side affected. Thus migration of a tooth always occurred in the direction away from the affected side. If the degenerative process affected the entire periodontal membrane, extrusion resulted. Figure 6 shows a photomicrograph of a mesio-distal section in lower premolar area, the radiogram of which is seen in Figure 2. Widening of the periodontal membrane on the distal is seen, the tooth migrating mesially.

Pocket formation is due to the apical proliferation of the epithelial attachment alongside the cementum of the tooth with final severance of the epithelium from root surface. The apical proliferation of

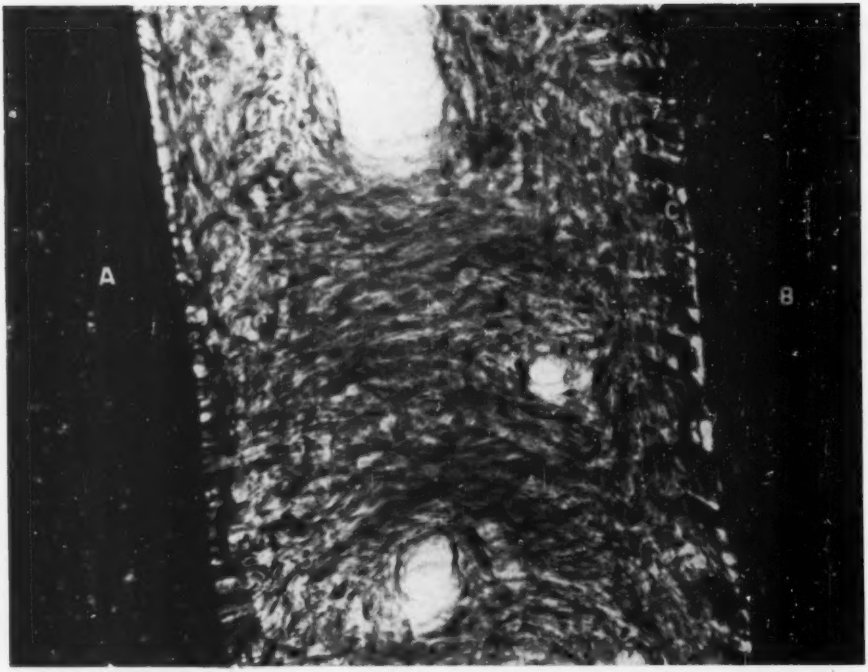

Fig. 3 This is a photomicrograph of the early changes in the periodontal membrane. Degenerated fibers are evident. A, cementum; B, bone; C, Sharpey's fibers. Wilder's stain, original, x500.

the epithelial attachment is possible only if there is loss of the attachment of the principal fibers of the periodontal membrane adjacent to the epithelium. As long as connective tissue fibers are attached to the root surface by a continuously forming cementum, no epithelium can migrate apically. Thus pocket formation was found only in those areas where the degenerative process of the periodontal membrane occurred in the marginal area. Study of pocket formation in these monkey jaws showed that pockets could be of all depths dependent upon the area of degeneration of the periodontal membrane. Therefore, deep pockets could arise almost spontaneously not being dependent upon a shallow pocket becoming deeper as time went on. Of course, this latter process did take place, but it did not account for the preliminary pocket formation. Since the alveolar crest was rarely resorbed initially, intra-bony pockets were the usual ones. Figure 7 shows a deep intrabony pocket on the mesial aspect of the maxillary first premolar, the radiogram of which can be seen in Figure 1.

That pocket formation was not associated with any inflammatory reaction was evident since in the many areas where gingival inflammation was present, no pockets of any depth were present. The crevices were found to be very shallow although the subepithelial tissue of the gingivae was completely filled with inflammatory cells. In one section studied, a foreign object was found penetrating the gingiva; although there was a severe inflammatory reaction, no apical migration of the epithelial attachment was seen.

The alveolar and supporting bone were of considerable interest in that the changes found were due to secondary factors rather than a primary loss. Resorption of bone was apparent in areas of migration of the teeth caused by a pressure atrophy such as seen in orthodontic movement. When a tooth moved in its position, the bone towards its movement showed evidence of resorption, (Fig. 6). Bone loss was also evident as a result of loss of functional stimuli where a breakdown of the periodontal membrane took

Fig. 4. This is a photomicrograph showing the degenerative changes of the periodontal membrane (A). B, bone; C, cementum. Wilder's stain, orignal x145.

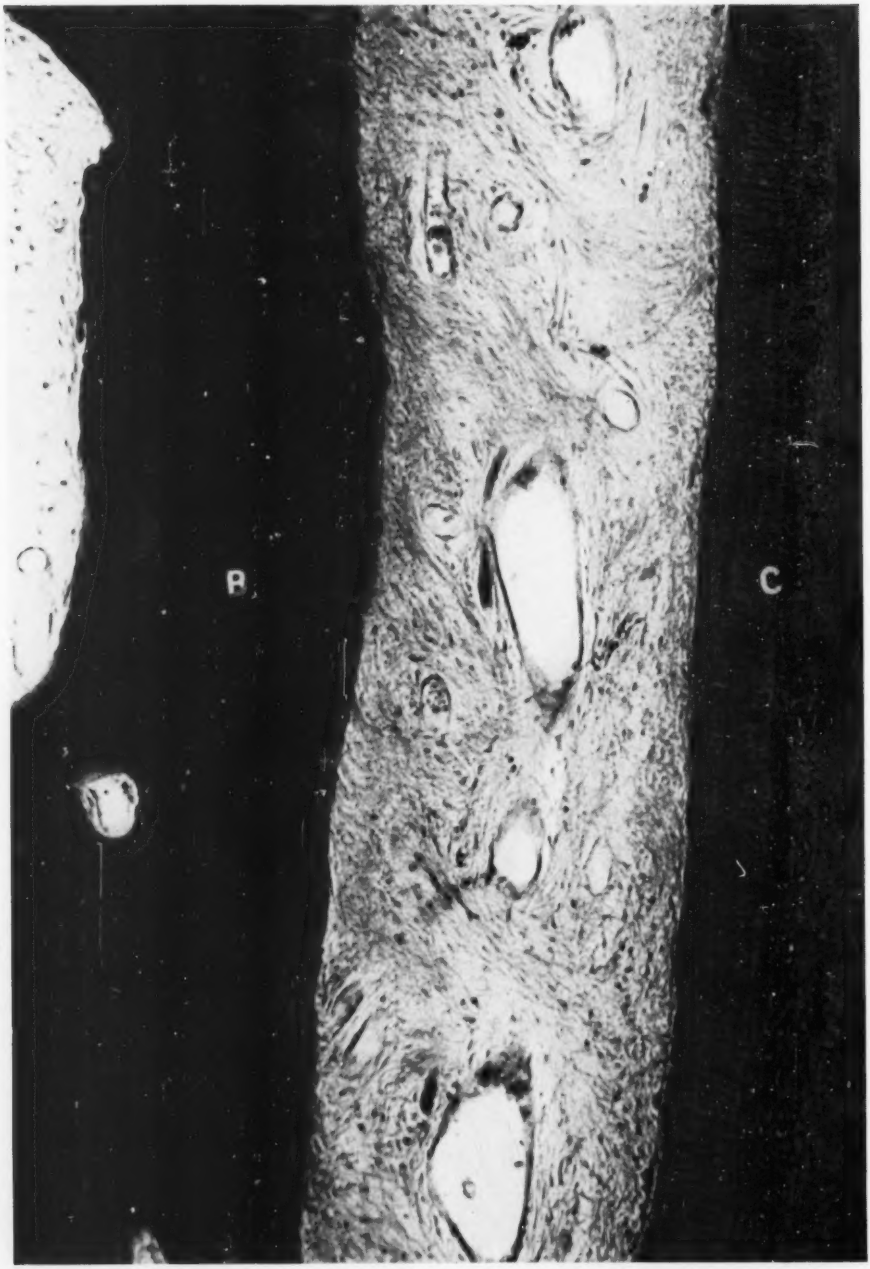

Fig. 5. This is a photomicrograph showing the complete loss of the principal fiber apparatus. The periodontal membrane is composed of a loose hyperemic, edematous connective tissue. B, bone; C, cementum. Wilder's stain, original x145.

Fig. 6 This is a photomicrograph of a lower premolar showing mesial migration. The radiogram can be seen in Fig. 2. Note that the interdental bone has been resorbed (A). Wilder's stain original x6.

place, the teeth being unable to transfer the force of occlusion through the periodontal membrane to the bone. Under these circumstances, it was the supporting bone which was affected. Also, secondary inflammatory conditions arising from the presence of pockets were the cause of bone loss.

Of considerable interest was the bone picture after the loss of teeth. Bone regeneration was active in the sockets; osteoblastic activity was noted and a rim of osteoid could be seen around newly formed bone. If this condition were primarily a bone disease, it is probable that no such activity of bone formation would be seen.

DISCUSSION

The clinical and histopathologic findings in these monkeys corroborate the observations of Gottlieb that periodontal disease can arise from a degenerative non-inflammatory process.

The histopathologic findings can be grouped into three stages and can be correlated to the sequence of clinical changes. In the first stage, degeneration of the principal fibers of the periodontal membrane occurs, resulting in that tissue becoming a loose hyperemic, edematous connective tissue. This loss of supporting apparatus allows for tooth movement, the tooth being pulled somewhat by the fibers of the opposite side and being pushed by the edematous tissue of the affected side. These changes account for the clinical symptoms of migration and loosening of the teeth. In the second stage in areas where the periodontal membrane adjacent to the epithelial attachment becomes affected, proliferation of the epithelial attachment alongside the cementum takes place. With subsequent separation of the epithelium from the cementum, pockets form. Thus all depths of pocket formation are encountered. The pockets are usually of the intra-bony type since the bony crest is not affected at this stage. In the third stage, inflammation due to irritation and infection from the pockets becomes marked. Bone becomes resorbed because of the tooth movement and also because of disuse; secondary inflammation conditions are also the cause of bone loss.

There are indications from many previous reports that systemic factors and toxic influences play an important part in the develop-

Fig. 7. This is a photomicrograph of a mesiodistal section of an upper premolar area, the radiogram of which is seen in Fig. 1. A deep intra-bony pocket can be seen at A. Wilder's stain, original x8.

ment of this disease. Gottlieb considered influenza to be the probable toxic causative factor in the first case he described. Toxic influences, produced by faulty digestive function, also have been thought to be among the responsible agents. In a case described by Gottlieb and Weinmann an unbalanced and deficient diet was thought to be the responsible agent.

Stafne and Austin reported a characteristic change in the dental radiograms of patients suffering from acrosclerosis and diffuse scleroderma, namely a widening of the periodontal space. This widening apparently occurred most often in association with acrosclerosis. Microscopic examination of one specimen revealed that the enlarged periodontal space was occupied by a loose, rather vascular connective tissue, the principal fiber apparatus being entirely obliterated. They believed that the thickening of the blood vessel walls suggested that the change in the periodontal membrane was probably brought about by the same circulatory disturbance which is a salient feature in acrosclerosis and diffuse scleroderma.

Thus from previous investigations, it seems that the cause or causes of periodontosis may lie in some nutritional deficiency or functional disease. The association of changes in the periodontal membrane with a connective tissue disease is most interesting and gives further light as to the changes seen in periodontosis.

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Making the General Practitioner More Periodontia-Minded

By JESSE L. WILLIAMS, D.D.S., *Jacksonville, Fla.*

"Why is Periodontal Disease So Generally Neglected in the General Practice of Dentistry and What Can the American Academy of Periodontology Do to Improve the Situation?" This was one subject discussed during the Luncheon Round Table at the 1946 meeting of the American Academy of Periodontology, and it has remained constantly on my mind ever since.

At the outset, it should be clearly understood that, except in cases of diffuse alveolar atrophy, periodontal disease begins with mild, indeed almost unnoticeable, gingivitis. In many instances it is due to unconscious but grave neglect on the part of both the patient and his dentist.

Is it not about time that dentists generally accept their responsibility in this matter and do something more about it, and not restrict their services to extractions, fillings, inlays, the making of fixed and removable bridges, and the insertion of partial and full dentures? The time has arrived when no patient should be able to say truthfully, "Doctor, I have had regular dental care all my life and have complied with all instruc-

tions given me by my dentist regarding my teeth and gums. How could my gums get into such a deplorable condition without it coming to my attention"? Surely it is high time that every dentist be taught the full value of the correct use of the dental probe in and about gum margins during an examination of the teeth.

Certainly, as an indispensable diagnostic aid, a well made full set of dental roentgenograms, including sixteen exposures, should be at hand and carefully interpreted. The routine roentgenogram studies, however, tell only part of the story. Where pockets are located, gutta-percha points should be inserted and more roentgenograms made to show the patient. Those pockets located on the labial, buccal and lingual surfaces are not demonstrated distinctly in the usual roentgenograms, as periodontists in particular and also dentists in general practice well know.

It is indeed past time that the patient be fully apprised of the seriously damaging effects of the loss of any normally placed permanent tooth, except the third molars, without proper replacement. It is not enough to suggest casually to the patient that a bridge could be inserted if desired. Instead, the dentist should tell him in an impressive manner about the injurious results and illustrate with models the ill effects and great damage the loss of the lower first molar, for example, can cause if it is not correctly replaced. There are some fourteen injurious results attributed to the neglect of proper replacement of this tooth.

What are some of the means by which dentists in general practice can be made more periodontia-minded? Chief among them are the following:

1. A clinic or lecture, repeated often, showing what can be accomplished in the treatment of periodontoclasia emphasizes the importance of this oral disease. This measure has already been nobly undertaken by a great many periodontists. But like John the Baptist, they seem to have been preaching in the wilderness. Even so, there is need for more such efforts.

2. The dental schools should be convinced of the necessity for more enthusiastic and thorough teaching of the subject of periodontia.

3. State boards of dental examiners should be urged to include an ample list of questions on periodontia in the examination given applicants for license to practice dentistry. A member of the Florida State Board of Dental Examiners assures me that these questions will be added to the examination given in this state.

4. An important step is convincing the laity that periodontal diseases can and actually are being cured by the proper treatment rendered by dentists and the coöperation of the patient.

5. The members of the medical profession should be informed of the need for treatment of periodontal diseases. They may well be impressed with the ability of many dentists to control and eradicate successfully the periodontal diseases, and also with the fact that pulpless abscessed teeth are not the only dental infections which are a serious detriment to the well-being of the patient.

6. The general practitioner of dentistry should be informed of the disappointment and resentment expressed by patients whose supporting tissues of the teeth have been allowed to get into a deplorable condition from disease and neglect. In most cases, the early signs are present. They should be recognized promptly, and treatment instituted at a time when correct curative and preventive measures can be most easily applied.

7. Adults and children should be taught effectual home care of the teeth. The correct toothbrush technic adapted for the individual patient makes possible effectual hygiene, which may be applied throughout life.

Editorials

What Is a Professional Journal?

[A REPETITION]

THIS question should have but one answer, yet, because of the continuing bewilderment about it, in the minds of dentists particularly, it deserves further discussion.

The function of a professional journal should be to inform, to inspire, and to further the education and advancement of the members of the profession it represents. Moreover, it should be consistent in contents, appearance, and objectives with the highest standards and ideals of the group sponsoring it. This is important because it is a part of the literature, it is the *record*, and in time the *history* of the profession.

This suggests the desirability of serious consideration before selling the most important pages in any dental journal—the front pages—to advertisers of dental materials and equipment. It will need much persuasion to convince an observant layman or an intelligent legislator that dentists are primarily interested in the scientific, educational, and dental health aspects of dentistry, if they have recently seen copies of the Journal which represents our largest and most important dental organization. In the last issue (34(2): Jan. 1947) one finds a total of 21 front pages (including the inside front cover page) devoted to advertising. The proportion of advertising to the Journal text in the December 1, 1946 issue, is nearly 2.3 (79 pages to 115 pages). The front pages of a professional journal should never be for sale. The fact that they are does not prove its desirability. It should not be operated for profit like the popular magazines but should have a better *raison d'etre*.

One popular magazine written for intelligent laymen and having but 135,000 subscribers prints approximately 100 pages of artioles, and in addition 7 pages of book reviews, several pages of letters to the editors and short biographies of its contributors, but much less than one-third of this total number of pages are used for advertising and these mostly for schools, colleges, and books. The subscription price is but five dollars. One wonders then why it is necessary to sell the front pages of the Journal of the American Dental Association. At the very least the page facing the first page of the text should be reserved. As it is now being used it makes an incongruous relationship between advertising and text. The value of a dental journal cannot be estimated by its income from advertising but by its usefulness to the members of the dental profession and indirectly to their patients.

Dentists who read do not look to their dental journals for entertainment, but for information regarding new ideas relating to dental practice, new developments in dental science and dental education, and other accomplishments

and problems of dentistry. The professional journal in dentistry should supply all this without the distraction and the excessive intrusion of advertisements.

Dentistry has "such a choice of difficulties" that this subject may seem a minor one, but is it?

An Explanation Not an Excuse

IT IS interesting to speculate on the probable cause of some long-standing situation that is complacently accepted by the majority of bystanders without anything being done about it. Not because it is unimportant, but because no one is sufficiently exercised over it to act. Such a situation is the tardy recognition of periodontal disease (especially periodontitis and periodontosis) and the habitual custom of advising extraction of all the teeth as the sole remedy. This procedure is recommended by many dentists and physicians without further consultation and regardless of the patient's past efforts to save his teeth.

While most dentists are dental caries conscious, too many are unaware of the destructive and insidious agencies often at work in and about the supporting tissues of the teeth, while a much greater number doubt the efficacy of any treatment other than extraction. It is the common regret of periodontists that the cases referred to them were not referred five, ten, or twenty years earlier. It is quite understandable that the individual operator might have preferences as to types of service, but if the colleges are teaching periodontia, as most of them claim to be, then diagnosis should be among the first phases for the student to understand and the dentist to practice. This should be made in the mouth and by use of radiograms and not by the latter alone. An ability to diagnose periodontal disease in the early stages would prevent the loss of many teeth.

It is pain that sends the majority of patients to the dentist. Cavities in the teeth can and do frequently ache and dentists know too well the fate of a tooth with an unfilled cavity. Unfortunately, very few diseases affecting the periodontium cause pain until well-advanced. For this reason patients do not visit the dentist very often for the relief of pain from periodontal disease. When they do it is many times too late and both (the dentist and the patient) believe it all happened recently, although it probably was many years in developing. Is it not possible that this one factor—pain, or rather the lack of it, explains also the lack of unfamiliarity with the early symptoms of many periodontal diseases?

If this is not the real explanation, we hope there is another very good one.

—G.R.S.

Nutrition and Dental Health

Selected and Reviewed

By DOROTHEA F. RADUSCH, D.D.S., B.A., M.S., *Minneapolis 2, Minnesota*

HEALTH OF REPATRIATED PRISONERS OF WAR FROM FAR EAST

MORGAN, N. J., WRIGHT, I. S. and VON RAVENSWAAG, A.:
J.A.M.A., 130:995, Apr. 13, 1946

A HEALTH SURVEY has been made of 4618 repatriated American prisoners of war from the Far East. An analysis was made of the results of prolonged malnutrition and mistreatment of these individuals. Striking deviations from normal include loss of weight, neurologic changes, a high incidence of pulmonary tuberculosis, muscular wasting, dermatologic manifestations, and psychologic changes.

After careful consideration by the examiners, it was felt that in a sense the survivors were a select group. They were largely seasoned soldiers, those with a "greater will to live" and thus were the ones who ate anything, however disagreeable, those who adjusted more readily. Furthermore, when examined, they had been for varying periods under highly therapeutic diets—this, too, influenced clinical findings.

In one Table, skin and mouth lesions are grouped. Of 4618 patients, one notes that only 37 had gingivitis with bleeding, only 196 atrophy of tongue papillae, with other evidences of change similarly uncommon.

The paragraph on dental findings, page 999, is quoted as follows:

> A detailed study of the dental examination forms has not been completed at this time, but as far as this study has progressed, no unusual pathologic conditions of the teeth were manifested. The number of carious teeth was less than that of the same age group living under normal conditions, which might be accounted for by the low amount of sugars in their dietary regimen. X-rays of the maxillas did not disclose any excessive bone destruction and the bony pattern was considered normal.

Since an incidence of only 37 cases of gingivitis in 4618 persons is vastly less than any practitioner of dentistry would find, or than has been reported by any survey, it would be desirable to know what criteria were used in examinations by the specialists assigned to the four debarkation hospitals on the east and west coasts.

In personal discussion, a dentist who was a prisoner of war during the entire period in the Philippines related that dental caries was almost non-existent but fractured teeth were not uncommon, probably due to the stones in the rice issued. However, soft tissue lesions of the mouth were the rule, not the exception.

Many surveys of various population groups have shown much higher incidence of gingivitis. Linghorne (*J. Canad. Med. A.* 54:106, 1946) for example, states that 20 per cent of non-selected R.C.A.F. personnel under 30 years of age had gingivitis to a degree readily detected by superficial examination of the oral cavity.

One wonders then if the almost non-existence of gingivitis is a true finding, or rather the result of the criteria used in making the examination.

GINGIVITIS AND ASCORBIC ACID LEVELS

The controversial question of the relationship of gingivitis to ascorbic acid nutrition has been reviewed (*J. Periodontology* 17:27, Jan. 1946). Since then, Linghorne and co-workers (*J. Canad. Med. A.* 54:106, 1946) have reported further study of the relationship between gingivitis, ascorbic acid intake, and blood levels. This report was reviewed in *Nutrition Reviews* 4:238, Aug. 1946, and is here quoted:

Linghorne and co-workers conclude that unless amounts of ascorbic acid in excess of 25 mg. per day are ingested there is demonstrable handicap to the gingival tissues. They were only able to demonstrate such handicap after pretreating the mouths of the subjects of the study so as to remove existing infection around the teeth; and they attribute their own early failure and the failure of others to find any benefits from increased ascorbic acid intakes to the pre-existing infection.

A total of 350 Royal Canadian Air Force personnel were the subjects of the several studies. In the first experiment it was found that administration of ascorbic acid or niacin alone, or a combination of Vitamin A, vitamin D, thiamine, riboflavin, niacin, and ascorbic acid for a period of five months resulted in no greater improvement in gingivitis than was observed in a control group. These subjects had received no special dental attention prior to institution of therapy.

In the final experiment, 150 subjects were given preliminary local treatment designed to remove all evidences of gingival inflammation, including careful removal of calculus and polishing of the teeth. The striking effects of this local treatment in the clearing of gingivitis initially present are illustrated by the excellent color photographs accompanying the paper. The 120 persons who were able to complete the entire eight months of the experiment were grouped as follows: 33 on a mean dietary ascorbic acid intake of 7.9 mg.; 30 on an average daily ascorbic acid intake of 22.3 mg. (the same diet as the former group but supplemented with raw cabbage); 31 with an intake of 77.9 mg. of ascorbic acid daily (the same basal diet plus a tablet containing 70 mg. of ascorbic acid); and 26 receiving the regular station diet with an average intake of 78.3 mg. Dietary control was maintained throughout and the serum levels of ascorbic acid served as a check on the intakes of the factor. Photographic records of the gingivae were made periodically under carefully standardized conditions. A biopsy of a dental papilla (of each case) was taken at the beginning and at the conclusion of the study. The dental examiners recorded bleeding tendency and tenderness employing a standardized technic which is described, and the degree of redness and swelling of the gums. At the end of the experimental period the incidence of these signs was increased in 45 per cent of the subjects receiving 7.9 mg. ascorbic acid per day, in 29 per cent with 22.3 mg. per day, in 18 per cent with 77.9 mg. per day, and in 5 per cent with the regular station diet.

The difference in development of gingivitis between the higher intake groups and either of the two lower intake groups was statistically significant. With the small groups involved it was not possible to be certain that the differences between the two lower or between the two higher intake groups were real. In fact, the failure to observe differences in plasma levels of ascorbic acid of the groups with intakes of 7.9 and 22.3 mg. respectively, may lead to some question that there developed a basic difference in the body stores of the vitamin in these two lower groups. It is, however, clear that body stores and serum levels were lower with both of these groups with restricted ascorbic acid intakes, and that there was a handicap to the gingival tissues.

Examination of the biopsy material revealed in no cases any specific changes which could be considered as characteristic of lack of ascorbic acid.

These studies demonstrate that provided existing gingivitis is eradicated, the development of marginal gingivitis is more likely to occur on a diet containing 8 to 22 mg. of ascorbic acid per day than on a diet high in ascorbic acid (approximately 78 mg.). The data do not fix the minimal level at which this effect is apparent but indicate that it is in excess of 22 mg. per day. Although the results cannot be interpreted to indicate that all of the marginal gingivitis within the general population is prefaced upon ascorbic acid deficiency, they suggest that even moderate ascorbic acid deficiency can play a role in the development of such gingivitis. They also demonstrate that once such gingivitis is established, an improved ascorbic acid nutrition for a few months will not eradicate the condition.

Bruxism*

Bruxism has been defined as, "the inaudible gnashing or audible grinding of teeth or cuspal interference during unsuspected movements of the mandible". It usually occurs during sleep but may also happen while awake.

Its frequency is greater than realized by the patient or the dentist. Patients are often unconscious of the habit until informed by someone of this fact. They may complain that their teeth are sore or ache, or their jaws are tired when they awake in the morning.

The causes may be varied but any physical discomfort or mental unrest would certainly be a contributing factor. It has been noted that children with pinworms often grind their teeth at night and when they get rid of the worms, this also disappears.

The recommended treatment was either a rubber splint or acrylic bite plane to be worn by the patient at night until the habit disappears. Gottlieb stated this may take six months, a year or even longer. A description of such a bite plane is published in the January, 1946 issue of this Journal. Cuspal interference should be corrected by grinding. It was also recommended that we take complete habit histories of all our patients, that we teach them the value and presence of the physiologic rest position as pointed out by Gottlieb, and that dentists should be more observant of evidences of abrasion and attrition of occlusal surfaces of teeth which can only be the result of grinding between meals.

With the increase in psychoneurosis, it is possible that bruxism will play an important role in our periodontal cases.

Psychosomatic medicine may aid in the further study of this subject and develop one recommended form of treatment—autosuggestion.

* By request, this Report of a Round Table Discussion at the last Annual Meeting of the A.A.P. was prepared by the *Chairman*, Dickson G. Bell, *San Francisco, California.*

The next "In Our Opinion" discussion will appear in the April, 1947 issue. Subject: "The Preparation for Certification in Periodontology."

BOOK REVIEW

Oral Medicine. By Lester W. Burket, M.D., D.D.S., Professor of Oral Medicine, The Thomas W. Evans Museum and Dental Institute School of Dentistry, University of Pennsylvania; Professor of Oral Medicine, Graduate School of Medicine, University of Pennsylvania. With a Section on Oral Aspects of Aviation Medicine by Major Alvin Goldhush, D.D.S., M.S., D.C., A.U.S. 350 Illustrations 60 in color. Philadelphia: J. P. Lippincott Co., 1946. 674 pp.

Burket's studies and writings during the past several years have been numerous and distinguished but *Oral Medicine* is his first complete book, and though an ambitious undertaking he has been equal to it.

The teeth and their supporting structures are an integral part of the human organism and it is obviously the dentist's province to consider the causes and effects of oral disease in relation to the general health of the patient. The oral mucosa often exhibits the first signs of systemic disease and the dentist must recognize the implications of such symptoms. Furthermore, this knowledge will enhance the value of his coöperation with the physician and simplify case management. "A dental consultation carries a definite responsibility in improving medical-dental relations."

The contents of the book are divided into eleven sections as follows: (1) General Considerations, (2) Fuso-spirochetal Infections, (3) Diseases of the Tongue and Dermatologic Disease of Dental Interest, Minor Stomatitides, (4) Diseases of the Organ Systems, (5) Nutritional Deficiencies and Diseases of Metabolism, (6) Diseases of the Blood and Blood-forming Organs, (7) The Specific Infectious Granulomata, (8) Focal Infection, Dental Pediatrics and Gerodontics, Hazards of Dental Practice, Dental Diseases of Occupational Origin, (9) Oral Aspects of Aviation Medicine, (10) Color Atlas, (11) Regional Diagnostic Index. These are again subdivided under thirty-three headings.

The completeness with which the entire field of medicine is covered may be judged by the fact that some 20 diseases of the Respiratory system are considered, 15 Cardio-vascular diseases, 17 of the Gastro-intestinal tract, and other regional diseases in proportion. In all, well over 100 conditions of the Organ Systems are discussed more or less fully. This reviewer would have been just as well satisfied if some of these had been omitted, especially those admittedly very rare.

Many useful suggestions are made for the taking of the patient's history. Because the dentist sees his patients rather routinely twice a year, he has an opportunity to obtain by tactful inquiry, significant information concerning their health even before subjective symptoms have sent them to the physician. The answers will be a key to the best procedure, whether it be to refer the patient to his physician, or use precautionary measures or prophylactic anti-biotic therapy. In cases of known subacute-bacterial endocarditis, penicillin or sulfur therapeusis is most important before and after dental surgery.

In making his oral examination Burket uses a practical diagram of the anatomic regions of the mouth for charting his findings. One could wish for a brief descriptive review of the normal picture of these parts as a background for the examination.

Laboratory procedures are frequently necessary to confirm a diagnosis; but while the author states that for the most part these studies can best be performed by a reputable hospital or similar laboratory, a full chapter (and an interesting one) is devoted to the technic of these procedures. It is indeed most important for the dentist to be able to understand the language of physician and technician if he is to hold his own in the consultation and to best serve the interests of the patient.

The oral aspects of diabetes are thoroughly discussed under the following headings: (1) The effect of diabetes on the periodontal structures and the oral mucosa, (2) The effect of diabetes on the incidence of dental caries and odontalgia, (3) The effect of oral foci of infection on diabetes mellitus, (4) Precautions for performing dental surgery on diabetic patients.

Addisons disease of the adrenal cortex is

a serious condition which frequently exhibits marked oral symptoms. The pigmentation characteristic of the disease is found on gums, cheek mucosa, palate, and tongue, as well as on the skin.

Adequate consideration is given to pregnancy gingivitis and other oral and dental conditions associated with sex hormone action and with the endocrines. In hyperparathyroidism there is general demineralization of the skeleton and this process in the alveolar bone results in extensive resorptions and cyst formation, with associated inflammatory changes in the gingivae and early loss of teeth. Hyper-parathyroidism is to be suspected whenever unusual or unexplainable areas of decalcification are found in the jaws.

The close relationship of oral pathology and various nephritides is recognized, some writers having estimated it as high as 65 per cent in the etiology of pyelitis.

In gastric ulcer, faulty masticatory function as well as infection from oral sources may aggravate or perpetuate the condition.

The role of infection in arthritis has been over-emphasized but in acute infectious arthritis, removal of suspicious teeth and correction of periodontal pathology is strongly indicated and the acute exacerbations which may follow the oral surgery will probably be of benefit.

In diseases of the eye where infection may be direct, radical elimination of possible oral foci is imperative. Dr. Burket considers periodontal disease the most significant of oral foci of infection. These lesions are more prevalent than periapical granulomas and the periodontium is highly vascular and rich in lymphatics, which favors dissemination of toxic products. His discussion of the entire concept of focal infection is conservative, interesting, and scholarly.

The dermatoses are important, the oral mucosa as well as the skin being of ectodermal origin. In treating leukoplakia the author has obtained marked improvement by requiring cessation from smoking, alcoholics, and highly seasoned foods; and in addition the patient is instructed to rub the lesions gently for 5 minutes twice daily with a soft cloth moistened with water and castile or other pure soap. The leathery areas soften and sometimes disappear under this treatment.

There is a good discussion of fuso-spirochetal infections. Smears are useful in showing progress of the disease and the effects of treatment. An interesting tabulation is given of the effects of various drugs on the oral flora.

The avitaminoses are adequately considered. Among other original observations, Burket thinks that the capillary fragility associated with periodontal disease may be an indication of lack of vitamin P and that it is this citrin content of citrous fruit juices which is responsible for their beneficial effects in some periodontal diseases.

Burket's discussion of herpetic stomatitis is the best we have seen. On the other hand the consideration of gingivitis, *per se* is brief and far from adequate in many respects, while the deeper lesions of the periodontium are not even mentioned. "Periodontoclasia" is apparently not in the author's vocabulary, nor is "periodontosis," which latter term presupposes a connection with the general health. Burket lists two forms of "gingivitis of primary bacterial causation" which we have not seen described elsewhere and one wonders whether they are indeed pathologic entities; the descriptions are not convincing in this regard. They are "streptococcal stomatitis" and "staphylococcal stomatitis."

A worthwhile chapter is devoted to the health hazards to which the dentist is exposed in his practice and the measures to counteract them.

Section X consists of 60 fine colored illustrations of various oral conditions with accompanying descriptive text; and the last section is a regional diagnostic index. Local etiologic factors of various oral conditions are listed in juxtaposition to their systemic background. These two chapters alone make this book a must for the periodontist.

There is included in the volume a timely and intelligent discussion of the Oral Aspects of Aviation Medicine by Major Alvin Goldhush.

The illustrations throughout the book are excellent and revealing. It is well organized and indexed so that any condition suspected may be found in it at a glance.

Altogether *Oral Medicine* is a valuable reference book.

—CELIA RICH